TRANSACTIONS

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MARCH - APRIL
1959

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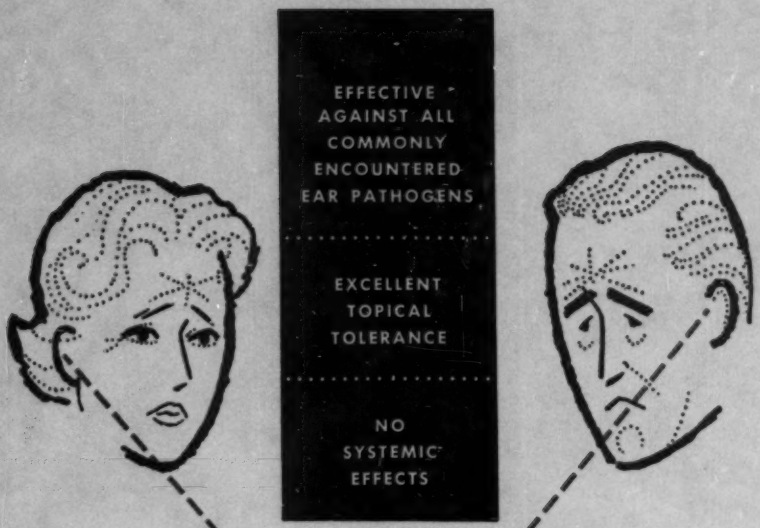

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American Academy of Ophthalmology and Otolaryngology

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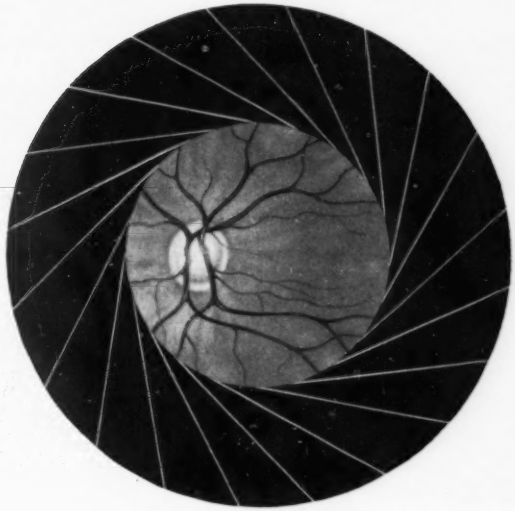

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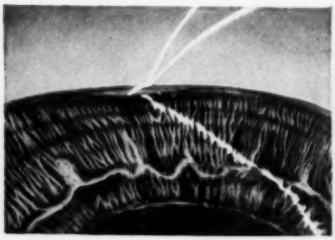

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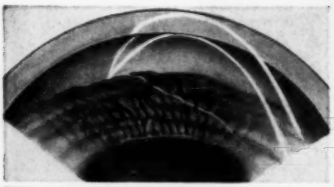

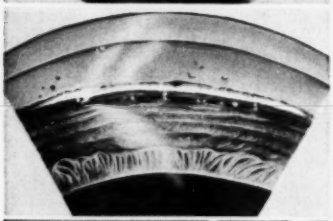

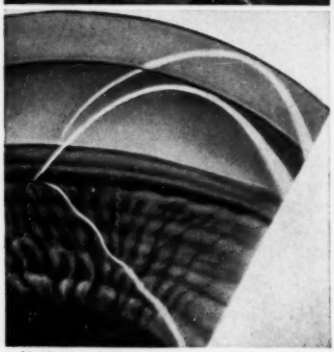

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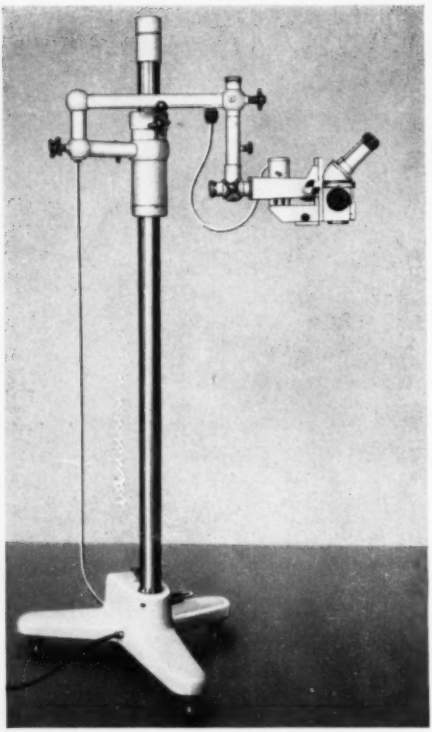

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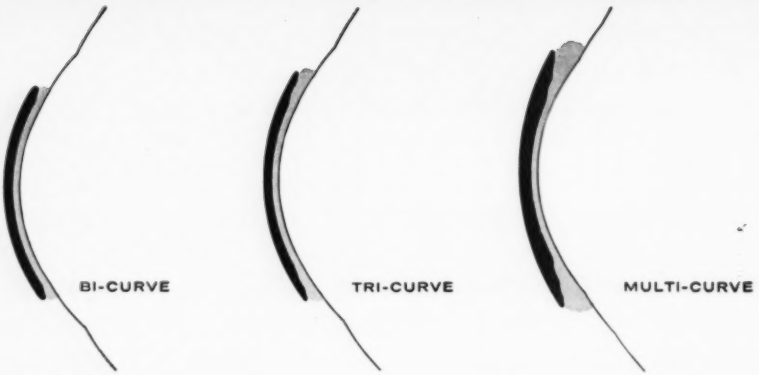

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2. Coyle, J.E.: Analysis of Blood and Vascular Factors in the Prophylaxis of Tonsillo-Adenoidal Hemorrhage, Laryngoscope, 10:1029 (Oct. 1957).

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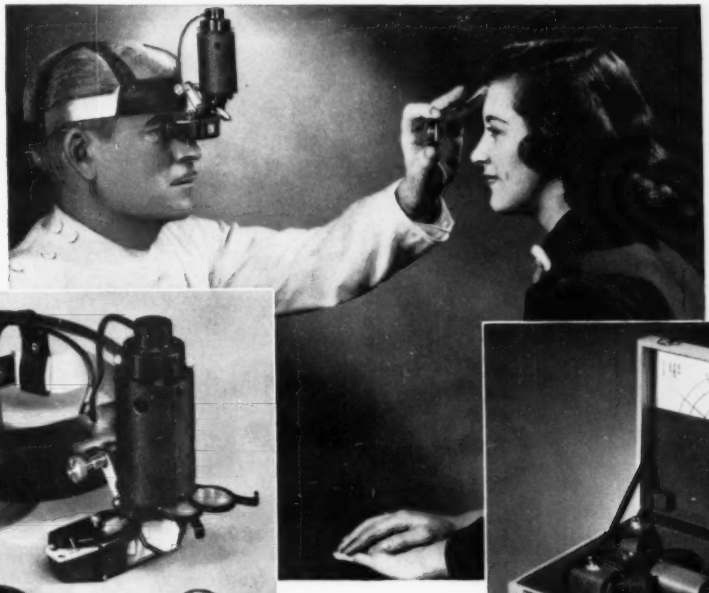

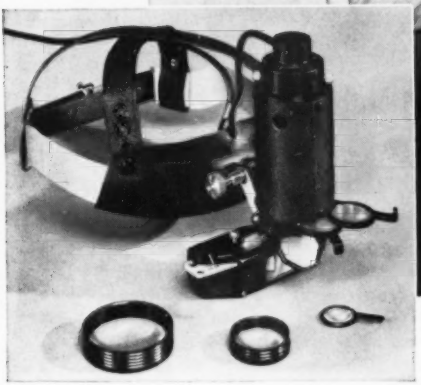

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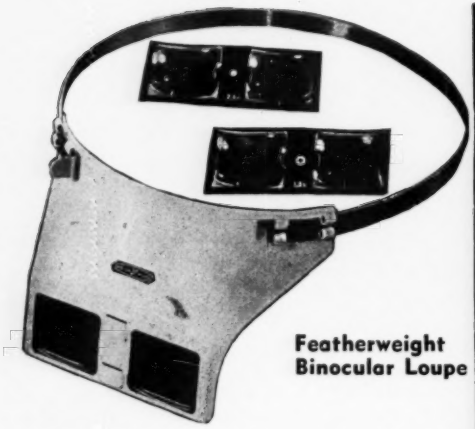

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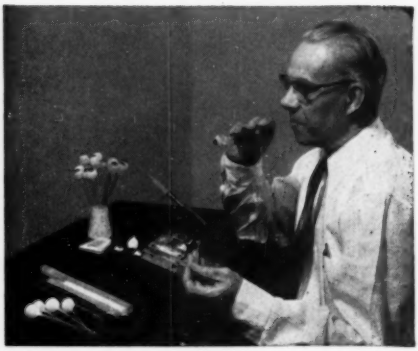

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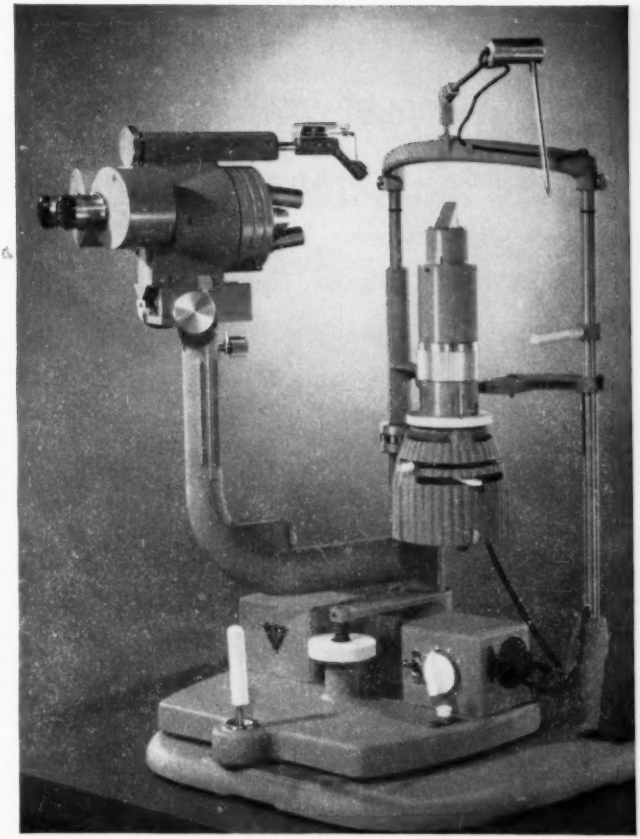

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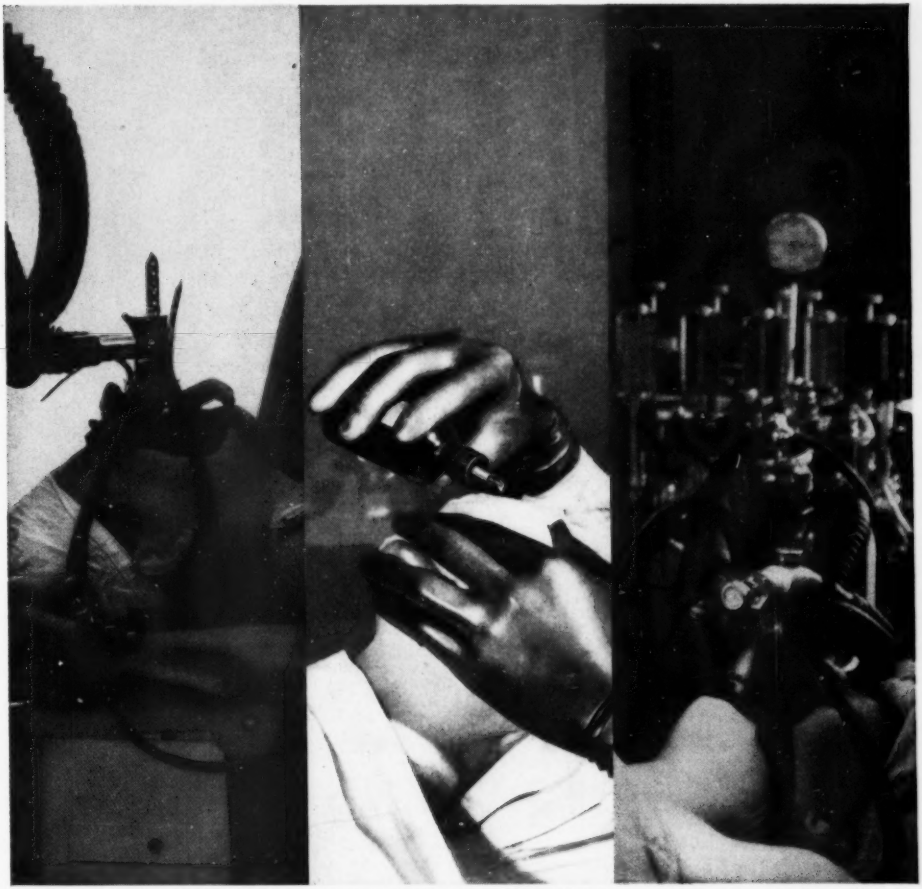

induction prior to ether—VINETHENE "... is more rapid and smooth than nitrous oxide and the excitement period is practically eliminated."[1] May be administered with closed technic.

short operative procedures—VINETHENE is easily administered by open-drop method. Induction and recovery are pleasant and rapid, muscular relaxation is good, and excitement or nausea is rarely observed.

supplement to nitrous oxide or ethylene—VINETHENE with ethylene-oxygen or nitrous oxide-oxygen complements these drugs and permits utilization of more oxygen. Ideal for brief surgical procedures.

1. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 2, New York, The Macmillan Company, 1955, p. 69.

supplied: In 10-cc., 25-cc., 50-cc., and 75-cc. bottles, each with adjustable plastic dropper cap.

MSD MERCK SHARP & DOHME DIVISION OF MERCK & CO., Inc., PHILADELPHIA 1, PA. VINETHENE is a trademark of Merck & Co., Inc.

A COMPLETE TREATMENT ROOM IN A SMALL AREA

The wall mounted treatment cabinet, treatment chair, cuspidor with suction shown above are contained in a 5 x 6 ft. space. Note the comparative absence of visible plumbing and electrical wires. Although this equipment may be used conveniently in a small area it need not be confined to this space. The floor type cabinet can be substituted for the wall mounted type. A number of room arrangements will be furnished on request.

WALL MOUNTED TREATMENT UNIT: Covered with stainless steel. Contains: air regulator with gauge, tubing, cut-off and air filter; nasopharyngoscope rheostat; push button; cautery transformer and rheostat; otoscope transformer and rheostat; transilluminator complete with rheostat, cord, handle lamps, antrum and frontal tips; seven metal drawers on fibre runners and one large drawer for towels; waste container; bakelite covered work table 21" x 12"; space for five spray bottles, twelve solution bottles, shelf for cotton jars; pocket for history and prescription cards; spray bottle heater. Presents an attractive appearance and will not tarnish and is easy to keep clean. Price complete without glassware $195.00. Without air but with electrical equipment $165.00. With air equipment only $150.00. Without either air or electrical equipment $115.00. Complete set glassware $41.00. Otoscope head, coiled cord and handle $29.50 complete.

FLOOR TYPE TREATMENT UNIT: Essentially the same as our wall mounted unit but designed for the physician who desires a movable floor type cabinet (glides are furnished if roll-around is not desired). May be obtained with built-in pressure. No. 501 DeVilbiss pump is used.

CHAIR: Can be placed in horizontal position, will not tip in this position; raised and lowered by large hand wheel 18" to 24"; paper covered headrest removable; chair locks with foot-pedal; bakelite arm-rests removable. Back locks rigidly in any position. Chair as shown but less light, in colors brown, green or black $150.00 (other colors available at $5.00 extra). By adding a footrest and turning handle chair can be used for turning tests. This additional equipment is easily and quickly removable. Add $30.00.

COMBINED SUCTION AND CUSPIDOR: Designed to eliminate the cost of installing a separate suction. The apparatus is 12" wide and projects 13¼" from the wall. The flushing rim and the suction is furnished with a vacuum breaker and should meet all plumbing requirements. Stop and trap not furnished. Available in ivory, green and white. Price $95.00.

INSTRUMENT TABLE: Table top size 11" x 17", heavy base of procelain enamel on casters, standard is chromium plated, table stainless steel. Single deck $34.00, double deck (as shown) $40.00.

EXAMINING LIGHTS: Chair mounted light, a flexible goose-neck lamp which can be moved up or down, this lamp may also be mounted on the wall as shown. Price $22.00. A telescopic light attached to the ceiling or may be attached to the base board in back of chair. Available in 4, 5, 6 and 7 ft. (extended). 4 ft. $16.50 (add one dollar per additional ft.).

LIGHT SHIELD: Takes an ordinary 100 watt lamp. Will not burst, spot can easily be rotated. $5.00.

PHYSICIAN'S STOOL: A soft upholstered stool with easy running ball bearing stem casters (shown). $20.00. Stool with back, $26.00. Colors as listed for treatment chairs.

All Prices are F.O.B. Los Angeles, Calif.

Surgical — Mechanical — Research, Inc.

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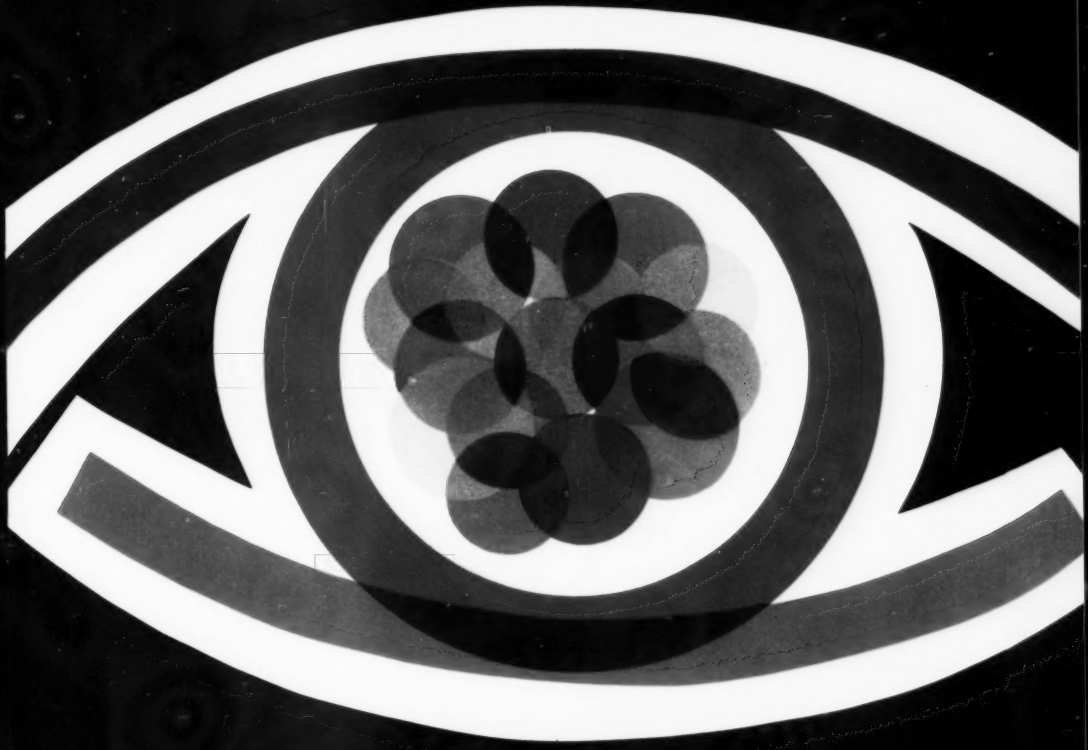
1901-1903-1905 Beverly Blvd. SMR Los Angeles 57, Calif.



the important factors
you look for in topical eye therapy

METIMYD

Ophthalmic Suspension

prednisolone acetate and sulfacetamide sodium

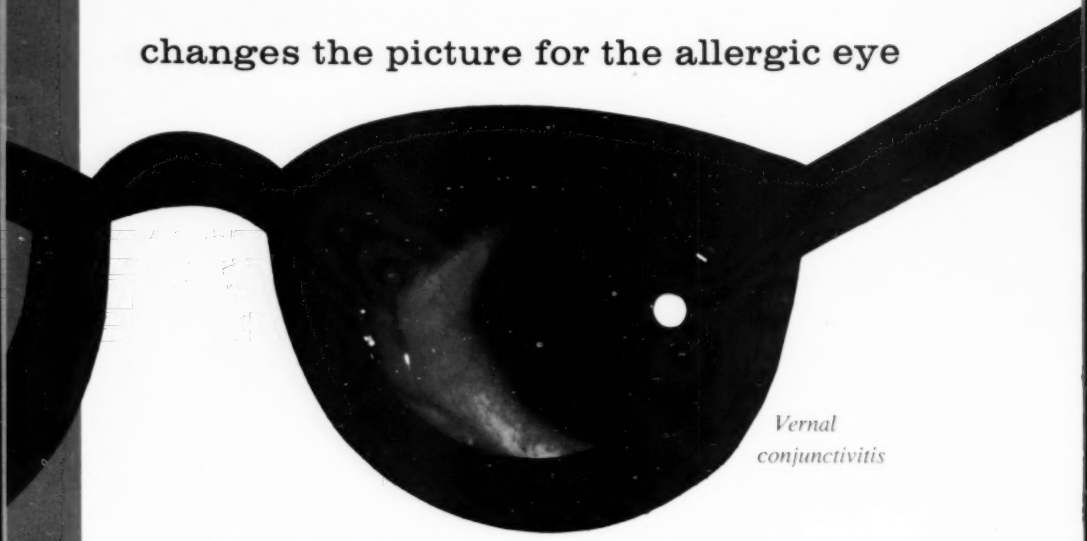
antibacterial
antiallergic
anti-inflammatory

Supplied: 5 cc. dropper bottle.

METIMYD,® brand of prednisolone acetate and sulfacetamide sodium.

Schering

changes the picture for the allergic eye



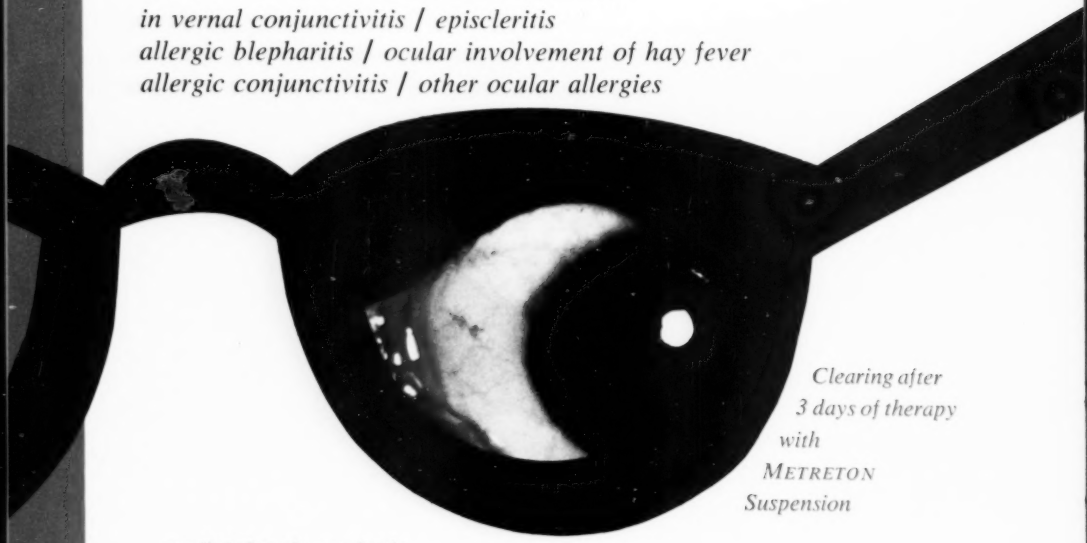
Vernal conjunctivitis

METRETON

Ophthalmic Suspension

combining METICORTELONE and CHLOR-TRIMETON

in vernal conjunctivitis / episcleritis
allergic blepharitis / ocular involvement of hay fever
allergic conjunctivitis / other ocular allergies



Clearing after 3 days of therapy with METRETON Suspension

supplied: 5 cc. dropper bottle.

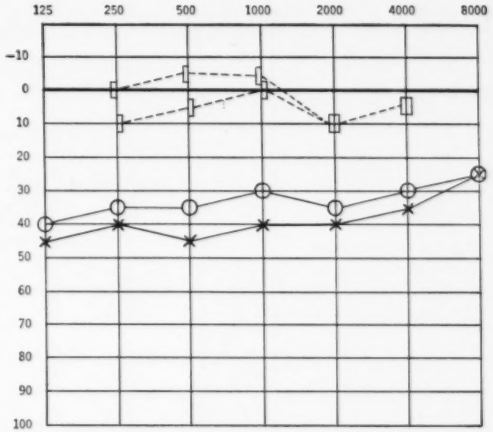
METRETON,® brand of corticoid-antihistamine compound.
METICORTELONE,® brand of prednisolone.
CHLOR-TRIMETON,® brand of chlorprophenpyridamine preparations.

Schering

SCHERING CORPORATION • BLOOMFIELD, NEW JERSEY

MS-J-119

Your Patient's Hearing Loss –and the Type Hearing Aid Needed!



If this were your patient's audiogram, which type of hearing aid would you recommend? It is taken from the case history of a 32-year-old bookkeeper whose hearing loss was diagnosed, at age 19, as otosclerosis, with little or no perceptive component. In consultation with his doctor, the patient decided against surgery.

Clinical findings: Ave. loss, R: 33 db, L: 42 db, B.B.A.: 33 db, SRT: 35 db, MCL: 55 db, TD: 100 db. Discrimination: L: 92%, R: 94%.

Prognosis: Patient should adapt readily to moderate gain hearing aid, air conduction type.

Recommendations: Any of these five Zenith Hearing Aids: Zenith "Citation" or "Challenger" Eyeglass Hearing Aid. At-the-Ear Zenith "Diplomat" with L-1 earphone, or "Ambassador." Moderate gain conventional Zenith Hearing Aid—"Crusader" model in "M" tone setting.

This report is but one of the many typical cases described in a helpful new book, prepared especially for the medical profession by the Hearing Aid Division of Zenith Radio Corporation.

Now available . . . a valuable reference book designed to help you recommend the right type of hearing aid for your patients. Besides the actual typical case history outlined above, Zenith's book, "Which Type Of Hearing Aid For Your Patient?", describes many other cases, and lists the appropriate Zenith Hearing Aid. Also contains a complete description of all types of Zenith Hearing Aids and their uses.

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HEARING AIDS

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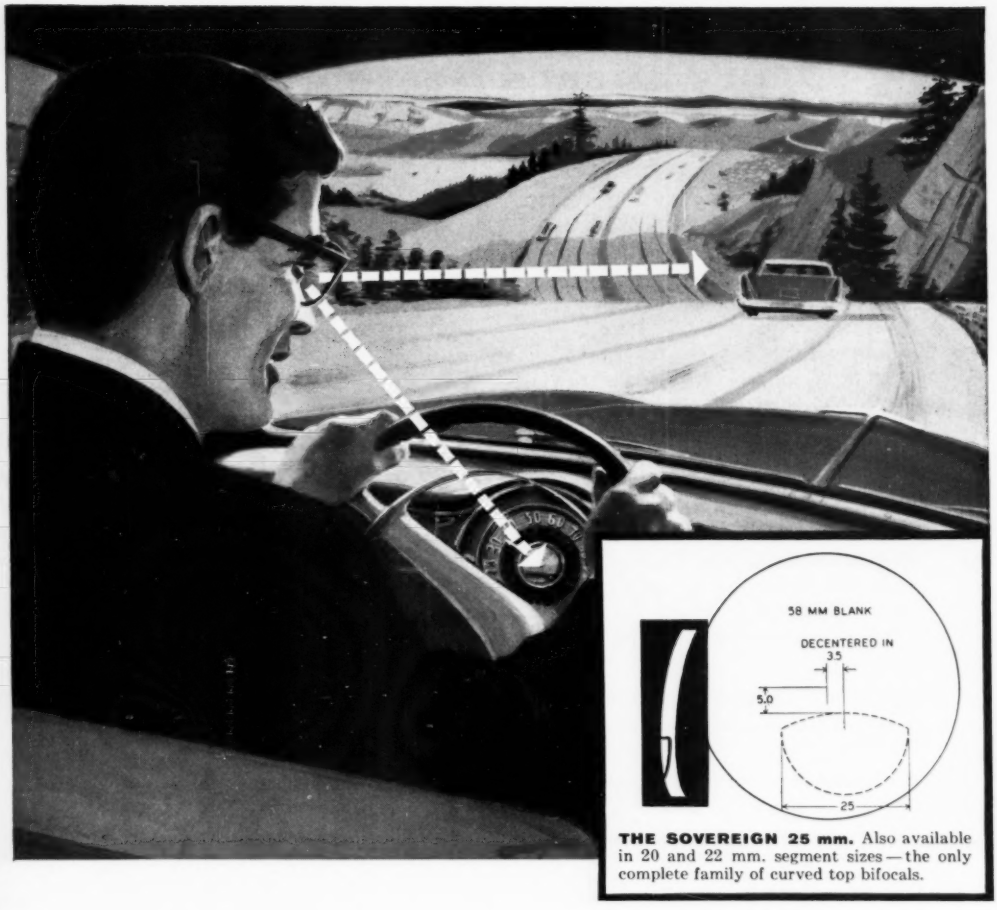

THE SOVEREIGN 25 mm. Also available in 20 and 22 mm. segment sizes—the only complete family of curved top bifocals.

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Indoors or outdoors, at work or at play, Tillyer Sovereign lenses put patients at ease.

The curved segment of a Sovereign lens follows the natural sweep of the eye and provides a more useful field of view. This curved top also helps scatter reflections. And its distinctive shape is instantly recognizable as a hallmark of quality.

Moreover, patients adjust quickly from distance to reading and back. And the Tillyer Principle of Corrected Curves gives undistorted vision clear to the edge. When a multifocal lens is called for, prescribe the Sovereign—a truly distinctive line of cut-off bifocals. Available through your AO Franchised Distributor or Local AO Branch.

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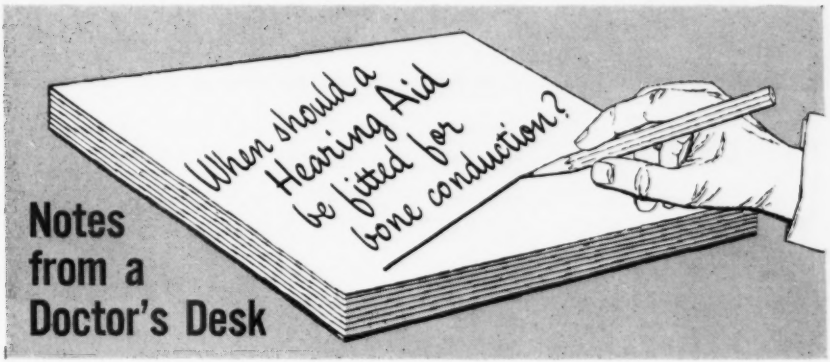
Since 1833... Better Vision for Better Living



Questions Asked by Doctors About Some of Maico's Products:

Hardly a day passes without the newspapers or magazines carrying an ad announcing a hearing aid "with nothing in the ear." Generally speaking, these advertisements are for a bone conduction type aid, with the receiver concealed in an eyeglass temple or held in place by a headband.

Bone Conduction Requirements

Perhaps the most frequently made error in hearing aid recommendations is the prescription of a bone conduction fitting because of the presence of conductive deafness and moderately increased bone conduction.

According to generally accepted practice, bone conduction should be recommended over air conduction only:

- *where a suppurative ear or other condition precludes the use of air conduction.*
- *where the patient insists on bone conduction and has 20 db. or better threshold for bone conduction than for air conduction in the speech range tones (500 through 2,000 cycles).*

Bone Conduction Fittings Under 1%

In the Audiology Centers established by the U. S. Army to handle hearing loss cases at the end of World War II, only 60 cases out of over 6,000 – less than one per cent – were fitted with bone conduction. They found that with a properly fitted earpiece, an aid with an air conduction receiver provided better discrimination and understanding. Furthermore, most hard of hearing persons prefer the comfort of an individually-molded earpiece to the headband arrangement necessary for good bone conduction.

MAICO Fits All Losses

The MAICO Dealer in your city has been thoroughly trained so as to understand the importance of correctly fitting a hearing aid to the specific requirements of the individual. With a complete line of precision-built hearing aids and hearing glasses, he is able to fit properly all types and degrees of hearing losses.

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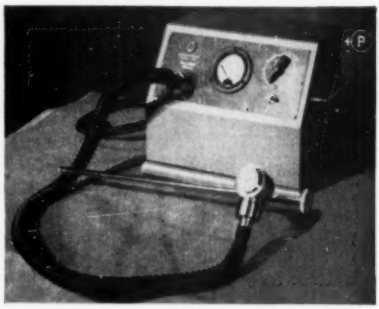
WHAT'S NEW *from* PILLING

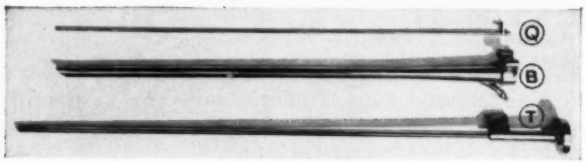
The Chevalier L. Jackson Fotobronchoscopic Unit for endobronchial photography

still or motion pictures / color or black and white

For diagnostic study, record keeping, clinical presentation and instruction...this set of instruments is the complete accessory unit needed for fine clinical picture-taking with your own camera.



bronchoscope 9mm x 40cm (B)
quartz rod for illumination (Q)
forward-viewing telescope (T)
luminator (L)
power unit (P)



Optics, certified by the Optical Institute, are imported from France.

The bronchoscope is of special design with light carrier canal inside the tube. This canal accommodates the standard Jackson light carrier and lamp, as well as the new quartz rod—thus the bronchoscope may be used in a routine fashion for regular procedures, or, with a quartz rod for photography.

The quartz rod is specially treated to transmit maximum light from the luminator to the distal end of the tube. These rods are encased in BRONCHALLOY, giving them a strength which belies the delicate design.

The forward-viewing telescope is complete with an anti-fogging device: an air jet across the face of the distal telescope lens assures constant clear vision. Adaptor rings are available to make proper contact between the proximal telescope eye-piece and the camera lens.

The luminator is the heart of the instrument . . . engineered so that its use is simple but very effective. The lamp in the luminator produces excellent light transmitted distally by the quartz rod. It is air-cooled by a large capacity pump housed in the power unit. Rheostat controls on the power unit provide variable light—four volts for regular bronchoscopy, up to 10½ volts for "over-illumination" of the lamp for the white light ideal for picture taking.

References:

Jackson, C.L.: New Instrumentarium for Peroral Endoscopy and Endoscopic Photography. LARYNGOSCOPE, *67*:777, (Aug.) 1957.

Fourestier, M., Gladu, A., and Vulmiere, J.: LA PRESSE MEDICALE, *60*:1292, (Oct.) 1952.

Dubois De Montreynaud, J.M., Edwards, Roger, J., and Gladu, Amédé J.: LARYNGOSCOPE, *66*:637, (June) 1956.

_____JOUR. BIOLOG. PHOTOG. ASSOC., *24*:45, (May) 1956.

The Jackson Fotobronchoscope is on display in Pilling's Philadelphia store and New York office. Write Philadelphia for further information and prices; send orders to Philadelphia.

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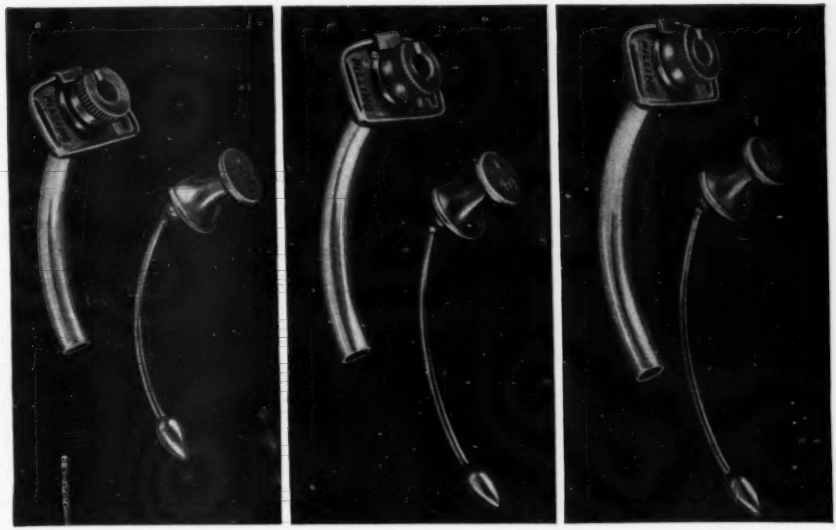
Designed by Paul H. Holinger, M.D., Chicago



ACTUAL SIZE: The size shown is #1—*l. to r.* 33 mm, 40 mm, and 46 mm long.

HOLINGER TRACHEA TUBES

Holinger Infant Trachea Tubes are designed to fit the smallest patient—down to four pounds—with as much comfort and convenience as possible. The unique curve of the tube has been developed as a result of X-ray studies of the position of the infant trachea and larynx—an arc of 65° rather than the usual 90° tube-to-plate alignment.

Infant Trachea Tubes available in 3 sizes, 3 lengths; please order by catalog number.

Br4270 Size 00—33 mm. long
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Br4275 Size 0—33 mm. long
Br4276 Size 0—40 mm. long
Br4277 Size 0—46 mm. long
Br4280 Size 1—33 mm. long
Br4281 Size 1—40 mm. long
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NOW AVAILABLE . . . a complete line of Holinger Trachea Tubes for adults. 8 sizes, each 3 lengths; please order by catalog number.

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Br4285-A Size 4—50 mm. long
Br4285-B Size 4—55 mm. long
Br4285-C Size 4—60 mm. long
Br4286-A Size 5—63 mm. long
Br4286-B Size 5—68 mm. long
Br4286-C Size 5—73 mm. long
Br4287-A Size 6—63 mm. long
Br4287-B Size 6—68 mm. long
Br4287-C Size 6—73 mm. long
Br4288-A Size 7—63 mm. long
Br4288-B Size 7—68 mm. long
Br4288-C Size 7—73 mm. long
Br4289-A Size 8—73 mm. long
Br4289-B Size 8—75 mm. long
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Holinger Laryngectomy Tubes available shortly.

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greater therapeutic efficacy

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CHLOROMYCETIN—*for effective, broad-spectrum therapy*

"...is effective against most gram-positive and gram-negative cocci and some gram-negative bacilli. It rarely produces dermatitis and resistant strains are uncommon."[1]

"...penetrates the noninflamed eye better than any other antibiotic, regardless of route of administration."[2]

POLYMYXIN B—*for control of gram-negative invaders*

"...gram-negative bacilli are being isolated with increasing frequency from the conjunctiva..."[3]

Polymyxin B "...is bactericidal against most gram-negative microorganisms..."[4]

HYDROCORTISONE—*for anti-inflammatory, antiallergic action*

"Cortisone, hydrocortisone and ACTH, by altering the inflammatory responses of the body, cause a decreased amount of scarring and vascularization."[5]

"Hydrocortisone is about twice as potent gram for gram as cortisone, even when given locally."[5]

INDICATIONS: For topical use in ocular infections due to organisms sensitive to Chloromycetin or polymyxin B.

ADMINISTRATION: Local application two to four times daily as required.

PACKAGING: OPHTHOCORT ointment contains 1% Chloromycetin® (chloramphenicol, Parke-Davis), 0.5% hydrocortisone acetate, and 5,000 units of polymyxin B sulfate per Gm., and is supplied in ⅛-oz. tubes.

REFERENCES: (1) Perkins, E. S.: *Practitioner* 178:575, 1957. (2) Queries and Minor Notes, *J.A.M.A.* 161:1032, 1956. (3) Smith, C. H.: *Eye, Ear, Nose & Throat Month.* 34:580, 1955. (4) Blakiston's New Gould Medical Dictionary, ed. 2, New York, McGraw-Hill Book Company, Inc., 1956, p. 945. (5) Ostler, H. B., & Braley, A. E.: *J. Iowa M. Soc.* 44:427, 1954.

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Procedure is shortened, and minimally traumatized grafts of even thickness and as large as permitted by the donor's eye are obtained. This result was difficult to achieve by older methods, especially with larger grafts.

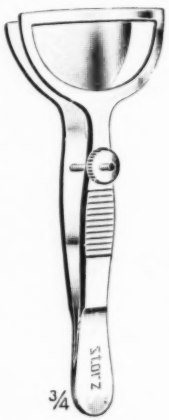
- Small enough to observe the donor eye for which it is used only.
- Edge of cutting blade visible through open roller guards, enabling surgeon to start and complete graft at exact margins.
- Uses small, disposable, double-edged razor blade. Simple mechanism.
- Shims are provided in thicknesses of 0.1, 0.2, 0.3, 0.4 and 0.5 mm., permitting grafts from 0.1 to 1.5 mm. thick. This method so accurately controls the thickness that two grafts of 6 mm. diameter have been removed from a **single donor eye.**

Instrument also useful for cutting buccal mucous grafts for plastic repair in cases of symblepharon or extensive scarring of the conjunctiva. The mucous membrane can be taken from the buccal surface of lip or cheek. It cuts the grafts so thin that they need no further thinning, which was impossible with previous instruments.

Head of instrument is Stainless Steel. Motor is rotary type, AC or DC, 110.

E-2950—Keratotome, CASTROVIEJO: head only with one dozen blades, Stainless Steel . **$150.00**
Foot Switch **14.50**
Norelco Motor **11.50**

Complete **$176.00**
E-2952—Extra Blades for E-2950, dozen **1.50**



E-2530 — Lip Plate, CASTROVIEJO: used in connection with the Electro-Keratotome for cutting of buccal mucous grafts **$17.50**

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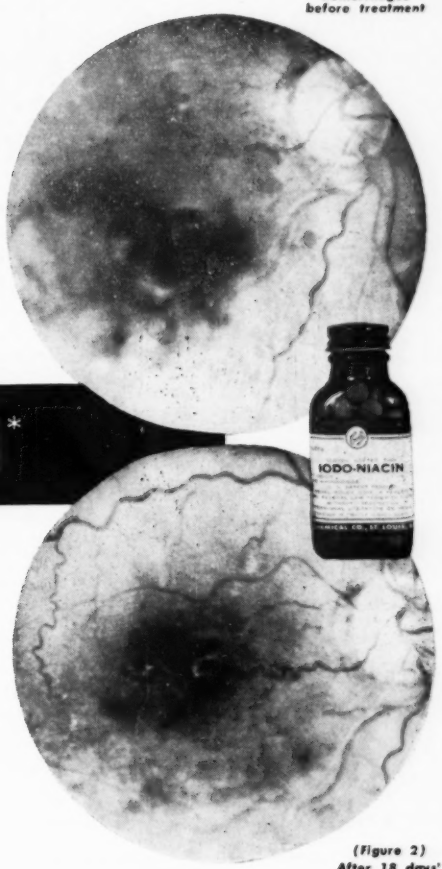
809 Lexington Ave.

retinal hemorrhages absorbed

with use of

IODO-NIACIN*

(Figure 1)
Retinal hemorrhages before treatment



(Figure 2)
After 18 days' treatment with Iodo-Niacin

As shown by retinal photography, rapid absorption of retinal hemorrhages follows use of IODO-NIACIN. These results have been established in a series of 22 cases, 12 of retinal and 10 of vitreous hemorrhages[1].

IODO-NIACIN Tablets contain potassium iodide 135 mg. (2¼ gr.) and niacinamide hydroiodide 25 mg. (⅜ gr.). The dosage used was 1 tablet three times a day. For greater effect this dosage may be doubled.

IODO-NIACIN may be administered in full dosage for a year or longer without any iodism or ill effect[2].

In emergencies, for rapid and intensive action, IODO-NIACIN Ampuls may be used intramuscularly or intravenously[3].

IODO-NIACIN Tablets are supplied in bottles of 100. Slosol coated pink. Ampuls 5 cc. in boxes of 10.

Cole CHEMICAL COMPANY
3721-27 Laclede Ave., St. Louis 8, Mo.

1. *Am. J. Ophth.* 42:771, 1956.
2. *Am. J. Digest. Dis.* 22:5, 1955.
3. *Med. Times* 84:741, 1956.

*U.S. Patent Pending

Write for professional samples and literature

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Gentlemen: Please send me professional literature and samples of IODO-NIACIN

M.D.

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make protection a part of every prescription

Every lens must withstand the impact of a ½" steel ball dropped from a height of more than 4 feet.
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HARDRx
SAFETY LENSES

BENSON'S HARDRx SAFETY LENSES!

Eyes worth correcting are worth protecting. All of your patients *deserve* maximum safety from dangerous and costly lens breakage. Benson's HARDRx safety lenses – ground to a formula-determined thickness and scientifically heat-treated – are toughened to resist impact.

To impress on your patients that you've prescribed the ultimate in lenses for them, the identifying tag shown at left is attached to each pair of genuine HARDRx lenses. Your patients will appreciate your thoughtfulness in prescribing this extra protection . . . and will tell their friends of your quality service.

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by ASH and RAUM

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"It is in the production of clear-cut, typical pictures in many magnifications that in most branches of medicine histopathology has done more than any other specialized technique to make the fundamentals of disease comprehensible. One of the reasons that diseases of the ear and nose are poorly understood is that routine pathologic material on the temporal bone and sinuses is difficult and expensive to obtain and process. Therefore it has not been done in most laboratories. "Never between two covers has so much basic pathology of the head and neck been compressed."
— (Edmund P. Fowler, Jr., M.D.)

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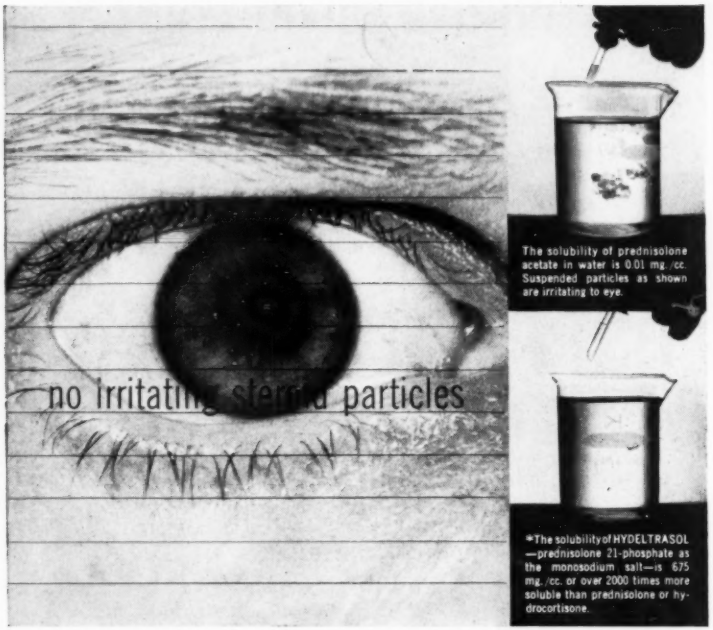
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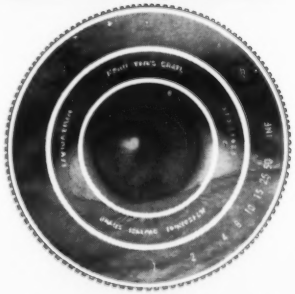

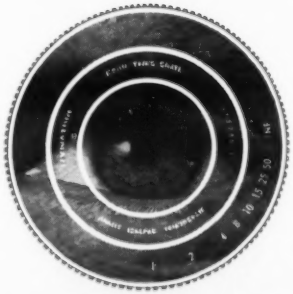

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ARTHUR LINKSZ, M.D.
NEW YORK, NEW YORK

I ARRIVED in this country in January 1939, and here I stand, not quite twenty years later, honored beyond my expectations, and certainly beyond my merits, delivering the Fifteenth Edward Jackson Memorial Lecture. These have been wonderful years—the best years of my life. I cannot help but feel humble and thankful at this, for me, so great moment.

The first recipients of this great honor were intimates and collaborators of Edward Jackson. The main subject of their lectures was the personality and the achievements of that great American ophthalmologist, his many faceted literary, organizational, educational and scientific work. Later lecturers often but invoked his name like one says grace at a festive occasion, presenting, in the main, some important aspect of their own scientific endeavor. I cannot follow either pattern. I can say little that is personal about Edward Jackson — though his work I know intimately—and as far as scientific achievement is concerned, I have precious little to offer. Experimental investigation, nowadays, is more and more in the hands of full-time medical investigators. Rare are the Duke-Elders, Friedenwalds and Goldmanns who excel in clinical medicine while still measuring up to the level of these professionals. Surely, I am not one of them. Experimental medical investigation requires time of which I, a practitioner, have little, and facilities, a research staff, an organization and, most of all, an endowment. Heaven knows, I never had much luck in joining, building or mustering any. There is, I feel, only one justification for my standing here today—my endeavors as a teacher of applied physiologic optics. Edward Jackson was, among many things, a teacher, especially a teacher of practical physiologic optics, and being an enthusiastic teacher, he used every occasion to impart knowledge. He attended hundreds of meetings. He spoke not only before the great national organizations; no local medical or ophthalmological society was too small for him. Reading in his papers one constantly has the feeling that their main purpose was didactic. He wanted to share his know-how; he wanted to make others better practitioners of the art he had first taught himself to master.

His papers, especially his editorials, are little masterpieces of clarity and readability, easily understandable. He was especially interested in the technique of the clinical examination and the tools used for this purpose. His most celebrated invention, the cross cylinder, is a tool to simplify and expedite clinical refraction. He was a shrewd observer of psychology, intensely interested in the reactions of both patient and doctor. A cross cylinder in the form of the Stokes lens was known before Jackson's time

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He noted that patients react poorly to the slow increases of cylinder power introduced by the turning of this lens and that they react faster to sudden changes. He modified the Stokes lens accordingly. He was not even aware that he had invented something new—a new and important tool. His first description of the cross cylinder (he first called it an "astigmic lens") is hidden in a paper in which he suggested the proper selection of lenses for the trial case. Only much later did he publish papers on the principle and the application of the cross cylinder. By the way, Jackson earned his first academic degree in engineering, and all his life he happily coupled the mind of an engineer with that of a biologist.

I came to a meeting of this great Academy for the first time in October 1941, in the third year of my life in America. I use the adjective "great" deliberately. I have been in many countries, to many meetings, and I still have to see anything to equal this institution which offers the concentrated effort of the best brains in our profession to better the work of their colleagues and, especially, of their juniors. In fact, it is this unique characteristic of our Academy which makes it the proper vehicle for an Edward Jackson Memorial Lecture. After Dr. Jackson's death, the heads of this organization searched for some means to honor his memory. Many suggestions were made and finally a memorial lecture was found the most appropriate. Dr. Lawrence T. Post, announcing the establishment of this lecture in an editorial in the TRANSACTIONS, especially emphasized that lecturers be "selecting subjects particularly appropriate to the young man," all because of Dr. Jackson's "special interest in the instruction of young men and his life-long endeavor to inculcate in them an interest in research."

As I have said, I attended the meeting of this Academy for the first time in October 1941. My great friend and benefactor, Dr. Walter Lancaster, who not long before had become my chief at the Dartmouth Eye Institute, invited me on a guided tour through the extended facilities of this meeting, which at that time were as strange to me as they have now become familiar. I remember every detail of those few minutes. We came down from Dr. Lancaster's room to this floor and walked toward the steps which lead to the gate of the commercial exhibits. Hardly had we passed the gate when (what a coincidence!) a little, very old, almost grotesque man, with old-fashioned wing collar, came toward us. Dr. Lancaster, tall, erect, handsome, himself nearly eighty years old, greeted him with unusual reverence, and then I heard him say: "Dr. Jackson, may I introduce Dr. Linksz." They exchanged a few words and parted. "Was this, by any chance, *Edward* Jackson, the inventor of the cross cylinder?" I asked. I confess, I was confused. I was never too good on dates but I knew that this invention by the great Edward Jackson went back to the 1880's, a time long before I was born. And I also knew that the editor of the *American Journal of Ophthalmology* (the only magazine in the English language to which the Eye Clinic in Budapest subscribed and which I had been reading regularly for some time, even before I came to this country) was also an Edward Jackson. But that the two could be the same never occurred to me; maybe they were father and son, I sometimes thought. On top of it, I could not even tell whether this little old man with the wing collar was *either* of the Jacksons I knew about. In any event, I am still one of those Jackson Memorial lecturers who can say: I knew Dr. Jackson personally. It was the only time I ever saw him.

The next Spring, that of the year 1942, was a period of great activity at the Dartmouth Eye Institute. Dr. Lancaster and Dr. Burian prepared for the meeting of the American Ophthalmological Society, at which aniseikonia was going to be discussed by no lesser man than Edward Jackson. It was at that meeting that Dr. Lancaster read one of his most precious papers, "A Reply to Criticisms of Aniseikonia." And, as it turned out, it was at that meeting that Dr. Jackson read his last paper.

There is a span of exactly sixty years between Jackson's first paper and his last. The first one was a comparative study of the action of several mydriatic alkaloids, read before the Pennsylvania State Medical Society in 1882. The last was the one I just mentioned. Its title was "Practical Importance of Aniseikonia." However, that meeting was not the last one Dr. Jackson attended. He still participated, and actively, in the October 1942 meeting of this, his beloved Academy. Two weeks later he died, in his eighty-seventh year. Like all those blessed and chosen by the Lord, he kept on going and working, in full possession of mental and physical capacities until the very end. His faithful Friday, Dr. William Crisp, gave a moving account of his last days in the leading paper of the *American Journal of Ophthalmology* of January 1943: "Edward Jackson, Student and Teacher." Dr. Crisp was properly chosen to deliver the first Edward Jackson Memorial Lecture; his other friend, the unforgettable Dr. Harry Gradle, gave the second.

The number of papers Jackson published within the span of the sixty years between 1882 and 1942 runs to more than six hundred. This information I have from Dr. William Bane of Denver, a friend and collaborator of Dr. Jackson's and owner of the best collection of his publications. Through the good offices of Dr. Ralph Danielson, also of Denver, I had access to this unique collection. It would have been almost impossible otherwise to become acquainted with the earlier work of Dr. Jackson. I want to express my thanks here publicly to both of these distinguished gentlemen whom I proudly call my friends and who, having been friends of Dr. Jackson, brought me as close to him as if I had known him intimately. The stories, anecdotes, sayings I collected listening to them would make a good lecture. I hope one day they can be prevailed upon to publish this precious lore.

Jackson's first and last papers, in an almost symbolic manner, show his lifelong preoccupation with problems of precise refraction, and in speaking today of "aniseikonia," I actually speak only of another factor adding to the precision of refraction, another factor which, in properly chosen cases, helps in relieving eyestrain.

The problem of eyestrain occupied Dr. Jackson almost from the beginning of his career. I am quite sure I cannot spend my time, and yours, better than by quoting what really are pearls of wisdom from his writings about this very subject. Here are two quotations from a lecture Dr. Jackson delivered at the Philadelphia Polyclinic in 1909. The first one reads as follows:

> Those who do not suffer from their ametropia do not come to us for relief. Those who do come do not endure with comfort the strain which an error of refraction imposes. The same error of refraction which will cause in one person no discomfort whatever will produce in another continuous aching of the eyes and head, in a third terrific paroxysms of sick headache, in a fourth conjunctivitis, in a fifth chronic inflammation of the lid margins, and in a sixth, disturbances of digestion and general nutrition.

What wise words, and how true also of aniseikonia, a condition which, to paraphrase Jackson, some "do not endure with comfort," while others do, and

"those who do not suffer from it do not come to us for relief."

And here is a second quotation:

> The low and moderate degrees of ametropia are more likely to cause chronic and obstinate headaches and other forms of nervous disorder than are the higher degrees of ametropia.

How true again and how well applicable, as I shall try to show, to the problem of aniseikonia. Jackson described in this paper the case of a physician suffering from "dizziness and nervous dispepsia," relieved by the constant wearing of

R: + .37 D sph ⌒ + .25 D cyl
L: + .25 D sph

This patient tried on two occasions within four years to leave off his distance correction and each time, Jackson writes, "there was suddenly increasing dizziness and disturbance of digestion which passed away with the renewed wearing of the correcting lenses."

Jackson was much intrigued by the problem of anisometropia as a source of eyestrain. I shall quote several passages from one of his still earlier papers on "The Correction of Anisometropia" (1896), because I think they are even more pertinent.

> Anisometropia, like ametropia, in general, makes trouble either by preventing perfect vision, or by making it difficult to attain, *so that the benefit of it is only secured at the cost of eye-strain.* And as with the various forms of ametropia, it is in the higher degrees and in later life, that it entails imperfect vision; *and in the lower degrees and earlier in life that it causes eye-strain.*

Ametropia, anisometropia and, we may add, aniseikonia behave very much in the same manner. It is (to paraphrase Jackson once more) in the higher degrees and in later life that aniseikonia entails imperfect *binocular* vision, but it is in the lower degrees and earlier in life that it causes eyestrain. Monocular aphakia is the most outstanding case in point. Spaeth, in a lecture before the Oxford Ophthalmological Congress in 1957, stated that in monocular aphakia when corrected by contact lens, aniseikonia of 5 and more per cent was found to be of no significance. He was certainly justified in making this statement. This large amount of aniseikonia (which by the very nature of things is usually encountered later in life) entails imperfect binocular vision only, not discomfort. As such, it is of no significance. But it would be erroneous to conclude, as several other authors implied, that lesser amounts of aniseikonia are of even lesser significance. It is, to use Jackson's wording, "in the lower degrees and earlier in life that it causes eye-strain."

Here is another quotation from Jackson's paper which stresses this very point.

> Where in one eye the ametropia is of such low degree as not to entail eye-strain, while the difference of refraction between the two eyes is so great as to preclude the focusing of both together upon the same object, *the patient will not suffer from eye-strain.* If however the second eye presents ametropia of low degree, distinctly differing in kind and degree from the ametropia of the first eye, symptoms of eye-strain are very liable to develop.

The case for the importance of aniseikonia could not have been stated in better terms. Obviously, in the first type of case, binocular vision will often be absent or, if present, rudimentary, and though the images in the two eyes, physically, are unequal, "the patient will not suffer from eye-strain." Aniseikonia, in this case, will be of no consequence. I give an example of such a case:

O.D. -6.00 D sph = -1.00 D cyl, axis 170
O.S. -.75 D sph
Right retinal image smaller by 5 per cent

This was a young woman who thought one *must* have binocular vision. She had

practiced eye exercises for a long time and I tried to dissuade her from having anything further done. She had a compulsive neurosis, as had her parents, about binocular vision and fusion, but she had no eyestrain.

Spaeth's, Constantine and McLean's, Goar's and my own cases of monocular aphakia corrected by contact lens and having a residual aniseikonia of 5 to 8 per cent also are good examples to prove the correctness of Jackson's statement, and still better examples are Jackson's two patients with unilateral aphakia corrected with regular spectacle lenses, who also had no discomfort. Jackson gave their refractive correction and visual acuity as follows:

(1) O.D. + 11.00 D sph ⌒ -1.37 D cyl, axis 68 = 24/20
O.S. -4.00 D sph ⌒ -1.12 D cyl, axis 80 = 20/40

(2) O.D. + 2.25 D sph ⌒ -.50 D cyl, axis 84 = 1.2
O.S. + 11.00 D sph ⌒ -2.50 D cyl, axis 125 = 1.3

and he added, "But no insurmountable difficulty from aniseikonia has been encountered after such correction."

People blessed with suppression have no trouble even if retinal image size difference runs in the neighborhood of 30 per cent, as it must have in Jackson's cases. On the contrary, symptoms of eyestrain are "very liable to develop" if the ametropia of both eyes is of low degree and that of the second eye "distinctly differing in kind and degree from the ametropia of the first eye." Patients with this type of anisometropia ordinarily are under the compulsion to fuse like emmetropes or iso-ametropes, since their retinal images are not sufficiently unequal. Whether we call the cause of their discomfort anisometropia or aniseikonia or even anisophoria does not really matter. They *do* have ocular discomfort. They *do* experience eyestrain. We can take Jackson's word for it. Here is an example; a young salesman with a long history of severe eyestrain, with the following correction:

O.D. -.50 D sph ⌒ -.50 D cyl, axis 85
O.S.+1.00 D sph ⌒ -.25 D cyl, axis 175

The image of the right eye was smaller by 1.5 per cent, and its correction brought relief. Or this other similar case:

O.D. -1.50 D sph
O.S.+.87 D sph ⌒ -.37 D cyl, axis 105

in which the image of the right eye was smaller by 2.75 per cent and only its correction made comfortable vision possible.

The proper limits for the full correction of anisometropia are, Jackson asserted, to some extent determined by the limits of the normal power of unequal accommodation, which he assumed to be 1.0 D, or at the most 1.5 D. However, Jackson correctly found this to be only a minor factor. Another important factor, possibly *the* most important factor, which determines whether or not binocular full correction in anisometropia is feasible is, according to Jackson, the prismatic effect of the peripheral portion of the correcting lenses. More than sixty years ago Jackson clearly noted what now is common knowledge — the fact that whenever the visual axis does not pass through the optical center of a correcting lens, there is an apparent displacement of the object looked at.

Let me quote from the same paper once more:

> If the lenses differ in kind or strength, the apparent displacements of the point looked at will correspondingly differ; and in order to bring the impression of that point upon the fovea in each eye, the visual axes will need to be turned in somewhat different directions. The power to turn the visual axes in different directions varies in different individuals but

is definitely limited in all. This limit is narrowest in the vertical direction. . . . Differences in the strength of the lenses used before the two eyes are commonly limited by the risk of producing vertical diplopia, or eye-strain *in the effort to avoid* vertical diplopia.

These wise words are worth stressing again. It is "the effort to avoid vertical diplopia" which causes eyestrain. This effort, of course, is not a conscious one. Fusion, if present, has the character of a compulsion. It is "the compulsion to fusion," if present and under undue stress, which causes eyestrain. Not the anisometropia as such, not the aniseikonia as such.

Once more I want to emphasize that this paper was written in 1896, four years before Bielschowsky and Hofman published their famous study, "On the Ocular Movements Which Are Not Subject to the Will," and thus established the fusional movements, the ocular movements we now generally call "vergence movements," as a separate entity. Jackson obviously referred to these same movements when he spoke of "the power to turn the visual axes in different directions." He clearly noted that this power is most limited in the vertical direction. He correctly stated that the difference in the necessary rotations of the eyes depends on the difference in lens power. He believed that in the vertical directions the eyes cannot usually overcome more than the differential prismatic effect of two diopters and he thought that this is generally the limit within which correction of anisometropia is tolerable.

Jackson recognized, by the way, that this problem of lens-induced forced vergence is related to the problem of unequal retinal images, which we now call aniseikonia. I quote again:

What is commonly spoken of as the difference in size of the retinal images, produced by correcting lenses of different strengths, is simply a special instance of this unequal prismatic action in the peripheral portion of unequal lenses; and the confusion and eye-strain associated therewith come about, not by reason of the mere inequality of size of images, but through the difference of position of what should be corresponding points to which attention is directed.

This relationship is clearly shown in the following diagrams. It is seen in figure 1 that if an emmetropic eye turns from looking at point A to gaze at a point B which is α degrees downward, it needs to turn α degrees. A hyperopic or aphakic eye, like the one shown in figure 2, when corrected with a spectacle lens receives a larger retinal image. Now if the aphakic eye wants to change its gaze to look from point A to point B, it has to turn $\alpha +$ degrees. Let me repeat: The retinal image of the panorama from A to B subtends in the phakic eye α degrees; to carry the gaze from A to B this eye has to turn α degrees. In the spectacle-corrected aphakic eye, the panorama from A to B covers the larger retinal area of $\alpha +$ angular value. Thus, if this eye turns its gaze from A to B, it has to rotate through $\alpha +$ degrees. The circumstance in which some section AB of the panorama covers a retinal sector of α degrees in one eye and $\alpha +$ degrees in the other eye is called *aniseikonia*. The circumstance that in looking from A to B one eye moves through α degrees while the other has to move through $\alpha +$ degrees, I have previously called lens-induced vergence. I shall hardly have to take any of your time proving the fact that the two circumstances are the same. If Jackson was correct in stating that lens-induced vergence movements are the principal limiting factor in successful full correction of anisometropia, he might just as well have said that this limit is set by aniseikonia, by what Lancaster called abnormal aniseikonia, by the inequality of images frequently en-

countered in eyes with corrected unequal errors of refraction. Our great master, Edward Jackson, contradicted himself in this matter. Even if in his last paper Jackson tried to prove that aniseikonia of some 30 per cent is, in an occasional case, well tolerated, his earlier writings on anisometropia and on eyestrain caused by anisometropia prove most convincingly that the differences in image size present a factor that is to be considered —but only moderate differences in image size. Those two cases of Jackson's prove little, one way or the other. Those two patients with monocular aphakia were comfortable not because aniseikonia is of no consequence but because of good suppression. (I might add that the ma-

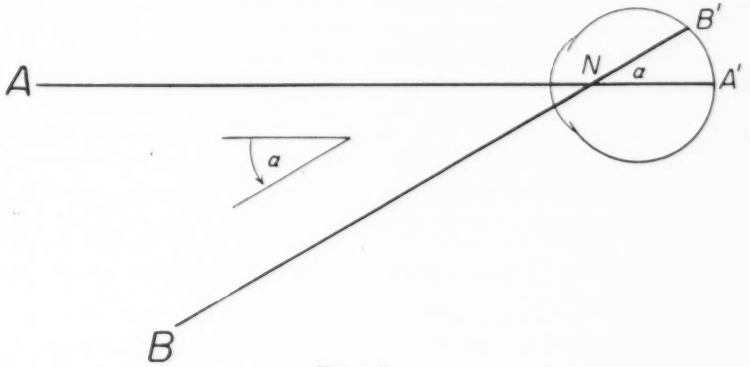


FIGURE 1.

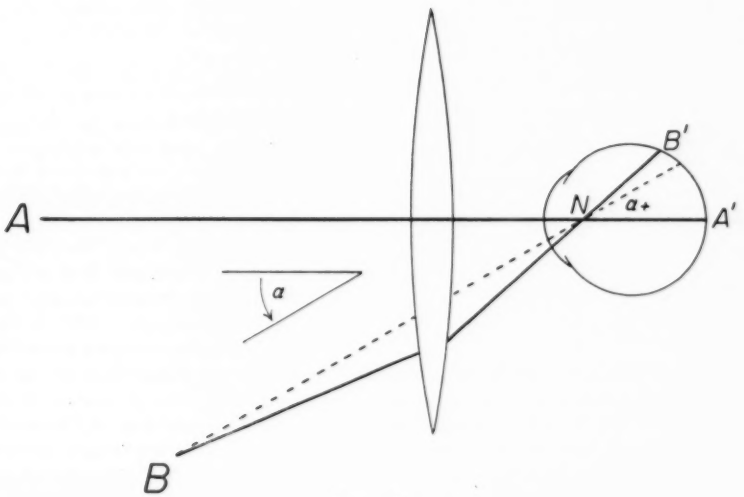


FIGURE 2.

jority of monocular aphakics who cannot wear regular spectacles do not offer a proof to the contrary, namely, that aniseikonia *is* a matter of consequence. Aniseikonia is the least of the problems faced by these people. Unfortunately, it would lead me too far to analyze, here and now, their problems.)

Jackson, of course, did not know at the time of his earlier writings that in order to have unequal retinal images in one's two eyes, it is not a prerequisite to have anisometropia and to wear a correcting lens, as the case may be, before one or both eyes. The studies of Ames and his collaborators at Dartmouth were still a matter of the future. It is, as we know today, possible to have unequality of retinal images with binocular emmetropia or iso-ametropia, though these cases are rare and the aniseikonia will hardly ever be of more than moderate degree. It is thus possible for a patient to be faced with the problem of forced vergence and eyestrain even without having anisometropia corrected by lenses of unequal strength. Jackson also did not know at that time that it is feasible to change retinal image size and retinal image position in any desired direction without influencing the eye's refraction. He did not yet have our size lenses at his disposal. Jackson assumed, and he assumed it correctly, that lens-induced vergence movements limit the feasibility of full correction in anisometropia and are, in this condition, the principal cause of eyestrain. Today, we can go further and add that size lenses eliminate just this need for lens-induced vergence movements, and with it at least one cause of eyestrain.

I should probably clarify this point. A change in the size of the retinal image, for better or worse, will generally also change the angle through which that eye need rotate in order to cover a certain section of the panorama. I shall quote no lesser authority than Ogle on this point:

> When a lens which has an angular magnification of 2 per cent relative to the center of rotation of the eye is placed before one eye, then, if the two eyes turn down to fixate a point 20 degrees below the axis of the lens, the eye with this lens before it must turn further by 2 per cent of 20 degrees, or 0.4 degree, or 0.7△.

Similarly, if the retinal image in one eye is larger for some reason by, say, 2 per cent in one eye than in the other eye, then, as Jackson clearly saw, unequal oculorotations of the just described magnitude will be necessary to cover equal sections of the panorama. However, in case a size lens of 2 per cent magnification is placed before the second eye, not only is the image size difference corrected (which Jackson might have thought unimportant) but the necessity of the forced vergence movements (which Jackson thought to be the cause of eyestrain) is also eliminated. If lens-induced forced vergence movements in anisometropia are the cause of eyestrain —and Jackson believed that they are— then size lenses are certainly the most logical means to eliminate them.

I must, at this point, discuss the relationship of these vergence movements to a condition known as anisophoria. Friedenwald, some years earlier and also at that memorable meeting in 1942, introduced the consideration of this entity into the controversy about aniseikonia. Lancaster clearly showed in the paper I have mentioned that the use of the term in this connection was, to say the least, unfortunate. "Phoria" is the term we all use to designate the relative position of the visual axes when fusion is *suspended*. If a person with fusion suspended shows, for instance, orthophoria looking straight ahead and a right hyperphoria of 4 prism diopters in the reading position, then this person re-

veals incomitant eye movements, possibly a weakness of the right superior oblique muscle; in short, he reveals anisophoria. If he has good binocular vision and proper vergence power, he might continue to have binocular vision and fusion in both positions. He might also have eyestrain due to forced vergence movements, which in this case has nothing to do with unequal corrections or aniseikonia. Let me repeat: In real anisophoria the eyes have a tendency to go through unequal rotations but are forced to equalize them for the maintenance of binocular fusion. In corrected anisometropia with no anisophoria present, conditions will be quite the opposite. Here the eyes have a tendency to go through equal rotations but are forced into unequal rotations (in other words, into a vergence movement) for the maintenance of the same goal. Under both sets of circumstances, eyestrain may ensue, but they are still not identical circumstances, and surely the forced misalignment of the visual axes under the second set of circumstances should not be called anisophoria.

One can, of course, have anisometropia *and* anisophoria. Lebensohn has stressed the importance of real anisophoria in clinical refraction. He was especially interested in the difference of vertical phoria in two selected positions, (1) looking at distance, and (2) with eyes depressed and converging for the task of reading. I have, unfortunately, no time to discuss this beautiful piece of work. I can mention only one of its most amazing aspects which is relevant to my topic. Lebensohn found that anisophoria will sometimes be of such degree and direction as to compensate for the undesirable differential prismatic effect of unequal corrections. Take the case of corrected right myopia of 4.00 diopters with emmetropia on the left side. Looking down to read through a portion of the lens which is, say, 8 mm. from its center, the myopic eye must turn less by 3.2 diopters than its fellow eye if binocular vision is to be maintained. This lens-induced vergence might be the cause of considerable eyestrain. Should this patient manifest a right hyperphoria of appropriate amount *only* when looking downward and not when looking straight, then the base-down effect of the minus lens in the lower field of gaze will just compensate for this, in this case of almost purposive anisophoria. No prism, no slabbing-off, no size lens will be necessary to equalize ocular rotations into this specific position.

A size lens is only one of the means of compensating for the forced vergence caused by unequal refractive correction or, to be more accurate, inequality of retinal images. A prism works in a similar manner, at least in one part of the field of gaze, in a specific direction. So will the slab-off method. The main difference is that a size lens influences the position of the retinal image and the extent of the ocular gaze movement in at least two directions (if it is of the meridional type) or in all directions (if it is of the over-all type), while a prism or a slab-off arrangement influences them in only one direction. If the essential difficulty is in the vertical meridian, especially in the reading position of gaze, a prism added to a bifocal segment or the slab-off arrangement might be sufficient and a cheaper solution. If the difficulty is caused by forced horizontal vergence or (what is the same) horizontal size difference, this type of optical correction will be useless. Here only size correction helps. To explain the essential similarity of action of the three seemingly so different principles of correction, some simple diagrams will help. These diagrams are, in fact, oversimplified and do not reproduce the complexities of the actual trajectories of rays, and for this I have to apologize. They will, I hope, serve well enough to clarify some points

Take again the two points, A and B (fig. 1), in visible space which at the nodal point of the simplified (so-called reduced) eye subtend an angle of α degrees. The separation of their images on the retina can also be said to be α degrees. The need for oculorotation to turn the gaze from A to B is also α degrees.

In the other eye (fig. 3), which carries a minus lens for correction of myopia, the nodal point ray from the same point B will reach the eye under a smaller angle α-, due to the prismatic effect so well known to Jackson. The retinal image of point B will be closer to the retinal image of point A. We generally say that the retinal image is smaller. And, as Jackson recognized, this eye has to turn only α- degrees to look from A to B. For the elimination of this difference in necessary version movements, of this forced vergence, there are, as I said, three methods available. Figure 4 shows the action of a so-called size lens. This lens gives the rays from B an added twist, counteracted by the corrective minus lens, so that the image finally reaches the retina under the desirable angle α. A prism which gives the ray from B the same desirable twist is shown in figure 5. Finally, the slab-off arrangement is shown (fig. 6). This gives the minus lens a second optical center in some more desirable position. Thus, the bending of the ray is eliminated and the image of B on the retina is in the desired location.

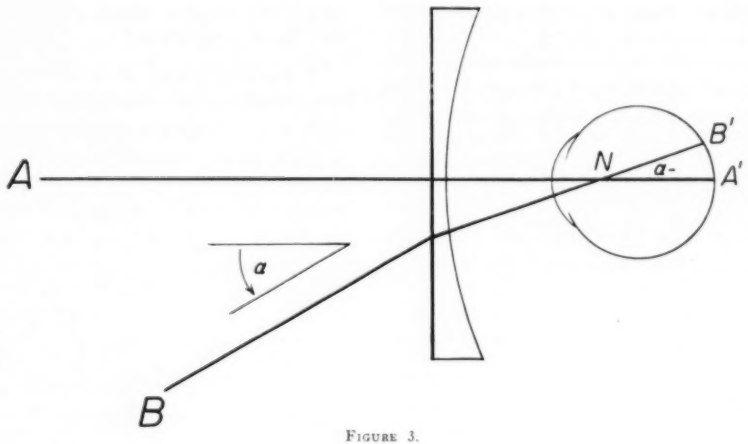


FIGURE 3.

The second solution works, of course, only in case bifocals are needed. Instead of a straight prism, one can, according to the procedure advocated especially by Lebensohn, use different bifocal segments: an Ultex-type segment in front of the emmetropic eye, or a flat-top segment in the concave lens, or a combination of both.

Here a justified question will arise: Why bother with eikonometry and iseikonic lenses if such seemingly simple measures help too? To this, the answer is twofold. First of all, one actually should not bother with size measurements and size lenses if such simple measures suffice. Second, one does not actually give

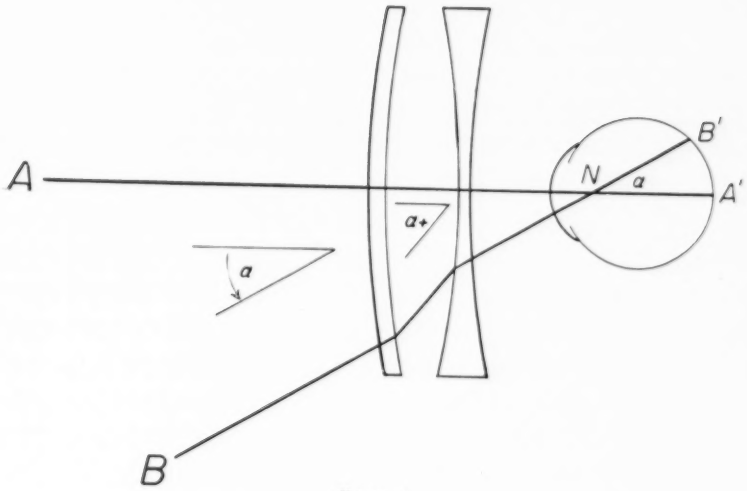
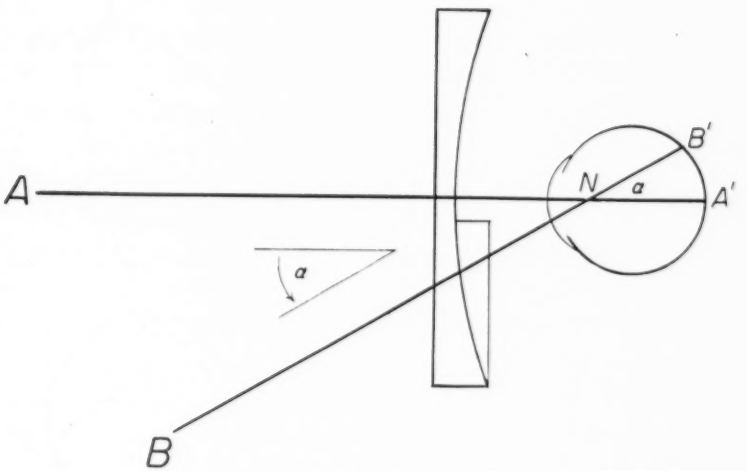


FIGURE 4.



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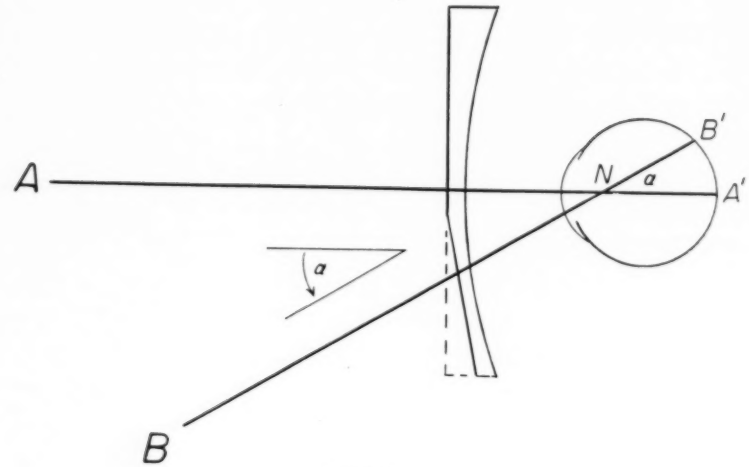

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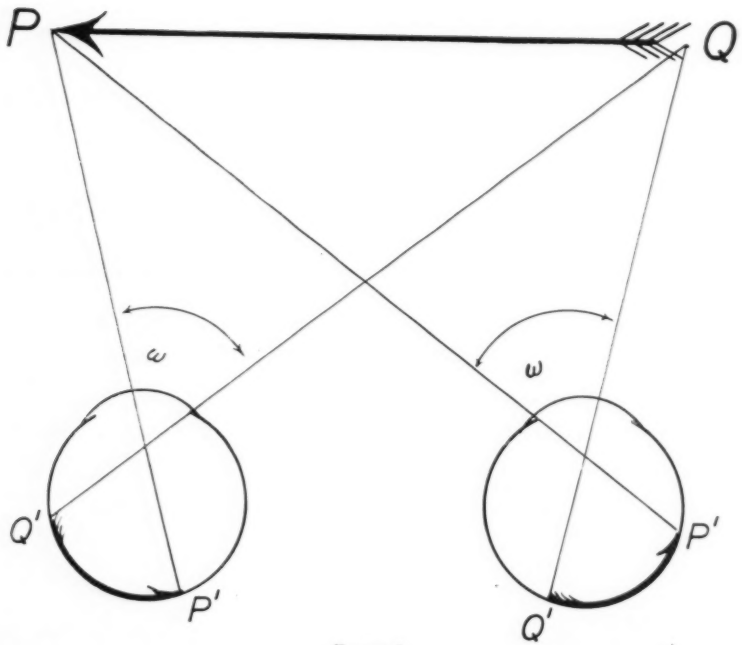

FIGURE 7.

full justice to the problem of aniseikonia if one completely identifies its effects with those of the forced ocular vergence movements, as Jackson did in his earlier papers. There is much more to aniseikonia, as the work of Ames and his collaborators has revealed. Forced vertical vergence movements might become a problem in reading, especially through bifocals, since eyes are not normally meant to carry out vertical vergence movements to the extent called for by considerable image inequality. But in the horizontal direction ocular vergence is part of our normal binocular equipment and aniseikonia is a normal occurrence. Our "normal" aniseikonia, as Lancaster called it, in the horizontal direction offers our clues of relative distance. There exists an intimate relationship between

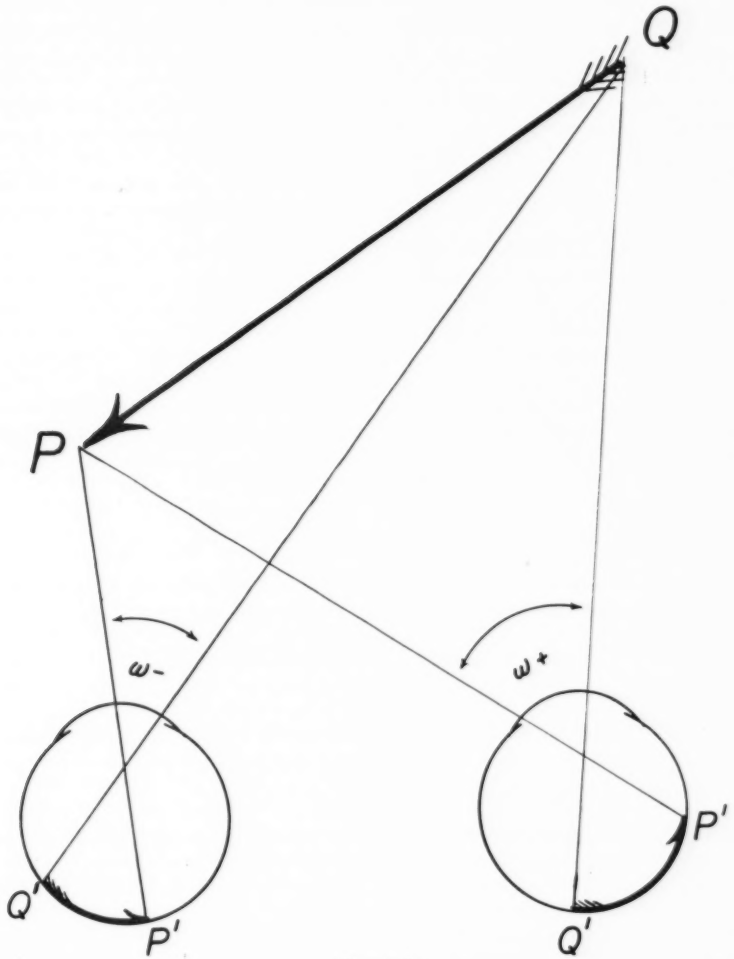

FIGURE 8.

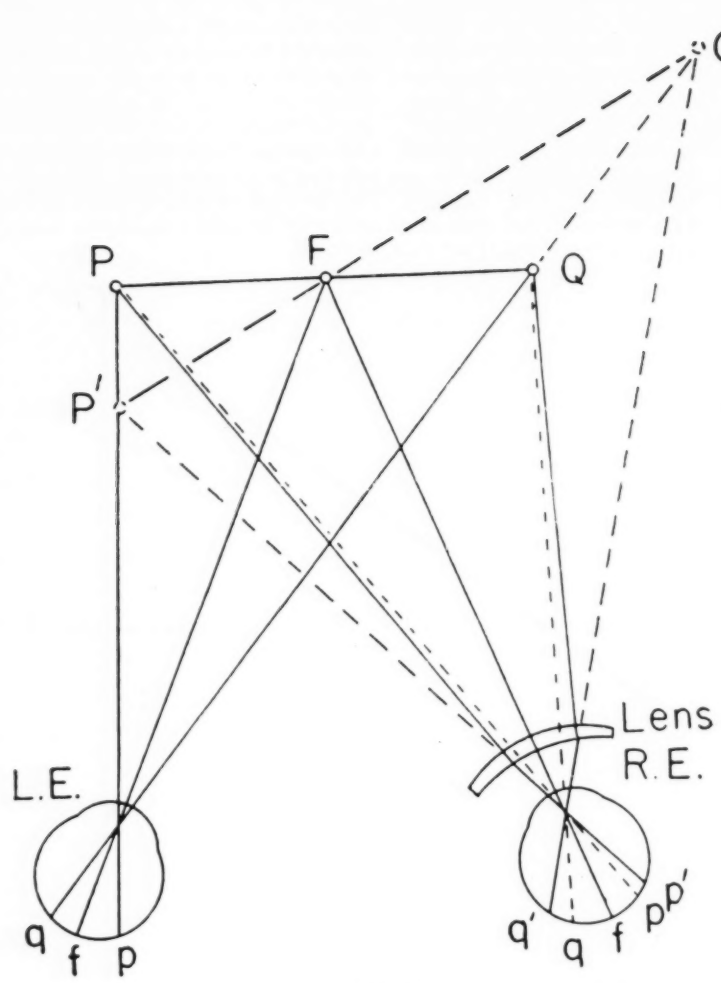

FIGURE 9.

horizontal image size difference, horizontal vergence movements, and spatial localization. In fact, these are the physiologic basis of stereopsis. "Abnormal" aniseikonia, if it exists in the horizontal direction, does much more than force the eyes into compensatory vergence movements. It falsifies our concept of spatial relationships.

Figure 7 shows the two eyes directed at some extended pattern *PQ* in the

frontal plane. If, as in this diagram, the pattern is symmetrical to the sagittal plane, then the retinal images in the two eyes are equal. And if the foveas turn from looking at *P* to looking at *Q*, then both eyes turn the same number of degrees. There is no need for any vergence movement.

In figure 8 the eyes are shown to look at the same pattern *PQ*. This, however, subtends an angle with the frontal plane and therefore its images in one eye (it happens to be the right eye) will be larger than in the other eye. And if the eyes turn from *P* to *Q*, the right eye will have to turn a greater number of degrees than the left. A vergence movement must occur in the horizontal, which certainly is not abnormal, and certainly is not inducive of eyestrain, which, in Professor Blank's opinion, is the very clue by which we tell that *Q* is farther from us than *P*. This is what Lancaster called "normal" aniseikonia.

Figure 9, from Ogle's book Researches in Binocular Vision, shows a magnifying lens placed before the right eye. The image becomes larger in the right eye and *Q* appears farther than *P*, while, in reality, both are at the same distance from the observer. If one has this type of image size difference, a wall might appear distorted though we know from experience that it is rectangular, the floor might appear slanting though our feet tell us that it is level. Jackson acknowledged that one might become aware of such distortions when wearing new eyeglasses, and he acknowledged at long last that aniseikonia is the cause of these disturbing experiences. But he strongly believed that the brain learns to re-interpret its clues. Of course it does.

Jackson believed that binocular spatial localization is altogether a learned function. Hering's views of innate spatial values were entirely strange to him. I am, of course, biased in the direction of Hering's views and wish I had time to discuss this important aspect of visual physiology on this occasion. But even if one assumes that binocular spatial localization is all learned, why should the brain be forced to re-interpret wrong clues when the clues can be made right without disturbing what to Jackson appeared to be the first concern—the accuracy of the refractive correction? I can, after many years of experience as a clinician, confidently make the statement that the correction of aniseikonia of small amounts, in the proper case, marks the difference between success and failure in the proper prescription of refractive correction. Why not avail ourselves of the additional method of improving our results in the proper cases?

The Manhattan Eye, Ear and Throat Hospital established its Aniseikonia Clinic in 1944 at the initiative of Dr. R. Townley Paton. Surely much credit is due to Dr. Paton for his imaginative leadership of this institution, and I personally must express deep gratitude for the opportunity given to me to do this work and grow with it. I ran that clinic for eleven years and during that time I saw a total of 2,666 cases in consultation. Of these, only a small part came through the hospital's refraction clinic—423 in all. A hospital refraction clinic is, in spite of the tremendous material available, usually not the right place to conduct research on problems of refraction, especially refinements of refraction. Most of the residents are uninterested in anything but surgery; we would need another Jackson, or another Lancaster, to impress upon the young generation that the study of vision and the correction of its defects is one of noblest endeavors, worthy of the attention of anybody who has had the title Doctor of Medicine bestowed on him. We need more eye doctors doing better refractions rather than lobbyists and public

relations experts to take care of people with vision problems. Most of the cases sent to the Aniseikonia Clinic were private patients sent by colleagues. The total of cases I saw represents an impressive material and its statistical evaluation might have been worth while. Unfortunately, I could not carry it out all by myself. I submitted several unsuccessful requests for grants. With the full-time help of a record librarian or a secretary and the part-time help of a statistician, much could have been accomplished. It would have been especially important to ask for follow-up notices from referring doctors or from patients, or both. The greatest defect in my material is poor follow-up. I had no personal jurisdiction over the majority of these patients—all those sent by colleagues. I acted only as consultant. I always sent a letter of evaluation to the referring doctor. Usually the letter contained some recommendation, often a suggested prescription. What happened to the patient I could only tell if the referring doctor cared to tell me or if the patient returned. At least half never did.

Since this method of evaluation turned out to be impossible, due to lack of time and help, I had to choose a different approach. I took the case histories of patients whose names started with the first and the last letters of the alphabet, 570 case histories in all, and perused them for the purpose of this lecture. This represented less than one fourth of the total material. And even of these a great number had to be discarded from further evaluation. Patients were often sent who had no place in an aniseikonia clinic, as, for instance, patients who had poor binocular vision, or none at all—even patients with obvious strabismus. They might have had considerable anisometropia (this was the reason for which many had been referred), they might, theoretically, have had unequal retinal images, but they certainly did not suffer from that strictly defined visual-functional disturbance in which binocular vision is present and compulsive and inequality of retinal images causes errors of spatial judgment and, first of all, eyestrain. Furthermore, there were many cases in which it became obvious to me that a better refraction or a more complete analysis of ocular motility was all that was needed, or that the case was one for muscle surgery. At one time I had clients of a New York reading institute sent to me. Many of these people were helped considerably through my efforts, but hardly any by a correction for aniseikonia. I think that aniseikonia as such is not a cause of reading difficulty. Therefore, all of these cases had to be eliminated. I also discounted most of those who were seen only once. Thus, from a total of 570, there finally remained a group of 265 in which a successful test for aniseikonia was carried out and who were either seen more than once or revealed some unusual or interesting features. There were another 164 cases available to me; these were my own patients in whom the refraction, ocular motility and binocular functions were tested by me and in whom the decision to carry out a test for aniseikonia was my own.

One of the interesting features in this limited survey is the existence of aniseikonia in patients who are emmetropic. As far as my clinic material is concerned, I always had to take the word of the referring physician, *his* criteria of emmetropia. One or the other patient might have been reported as an emmetrope simply because he or she had 20/20 vision without glasses in each eye. But even in my private material there are cases in which, with the most meticulous of subjective tests, especially the Lancaster-Regan dial, I could detect no astigmatism or spherical error. And there were also cases showing aniseikonia in which the error of refraction was iden-

tical in both eyes. In order to avoid any misunderstanding I want to make it clear that patients with the following correction:

O.D. +1.00 D sph ⊂ -2.00 D cyl, axis 135
O.S. +1.00 D sph ⊂ -2.00 D cyl, axis 45

cannot be considered to have identical refractive error in the two eyes, at least from the point of view of the aniseikonic clinician. Burian and Ogle emphasized this quite some time ago. Such a case must be considered to have a difference in refraction of two diopters in both the 45 and 135 meridians and can be expected to reveal considerable image size difference in these oblique meridians. Cases with this type of refractive error:

O.D. +1.00 D sph ⊂ -.50 D cyl, axis 180
O.S. +1.00 D sph ⊂ -.50 D cyl, axis 90

are also not iso-ametropes. Several patients with this type of refractive error were found to have considerable visual difficulty and eyestrain. They have definite, easily measurable aniseikonia.

Among my own 164 patients tested for aniseikonia, there were 30 cases with no error in refraction, or no difference in refractive error in the two eyes; there were 41 cases in which the greatest difference in any meridian was 0.12 to 0.37 diopters, and 43 in which the difference was 0.50 to 1.00 diopters. Thirty-two patients had a maximum difference of 1.12 to 2.00 diopters, and in 18, the difference between the refractive errors of the two eyes was over 2.00 diopters.

It is amazing to see that, even in my practice, in which by the very nature of my specialized interest there is a greater percentage of patients with eyestrain, headaches or reading difficulty than in the usual ophthalmologist's practice, the number of patients with a difference in refraction of more than 2.00 diopters *and* in need of correction for aniseikonia is so small. Again I must remind you of Jackson's words of wisdom, that only people with lesser amounts of anisometropia have eyestrain; those with larger amounts just don't have the compulsion to binocular vision. If sent to the aniseikonic clinician, they seem to have been referred for no justifiable reason. They might have aniseikonia in the literal sense—unequal retinal images—but they do not *suffer* from aniseikonia, that derangement of binocular vision which is the topic of this discussion, because they have no, or only rudimentary, binocular vision.

TABLE I

OVER-ALL IMAGE SIZE DIFFERENCE IN RELATION TO REFRACTIVE DIFFERENCE IN DIOPTERS IN 164 PRIVATE PATIENTS

REFRACTIVE DIFFERENCE IN DIOPTERS	NO. OF CASES	PATIENTS WITH OVER-ALL IMAGE SIZE DIFFERENCE					
		0.50—0.75%		1.00—1.75%		2.00% AND OVER	
		NO.	%	NO.	%	NO.	%
0.0-0.37	71	20	28	18	25	1	1
0.50-1.00	43	8	18	7	16	1	2
1.12-2.00	32	5	16	13	47	3	9
>2.00	18	1	6	5	28	5	28

Table I shows some of the data of my office material. (I shall restrict my discussion to those cases that show over-all size difference.) The patients who show a difference of not more than one diopter between the two eyes belong in the same group, statistically. Patients with anisometropia greater than one diopter again are a somewhat uniform group, though there is some difference. In the group in which the difference in refraction between the two eyes is more than one but less than two diopters, over-all image size differences of 1 per cent to 1.75 per cent prevail, while in the group in which the difference in refraction is greater than two diopters, a significant number have aniseikonia greater than 2 per cent.

Patients with meridional size difference only do not show such a definite trend. There is not a sufficient number with over-all and meridional size difference in my material to warrant any conclusions. Rare also are the patients in which the image of one eye is smaller than that of the other eye in one meridian and larger in the opposite meridian. I have not tabulated these values for this report.

Among the hospital cases which I had a chance to analyze for this review (table II), there are a sufficient number, 32 of the 265, in which the referring doctors reported no refractive error. I thought it worth while to separate them for this study. Four of them, or 12 per cent, had an over-all image size difference greater than 1 per cent. Amazingly, the largest category in this group of 265 is made up of cases in which the difference in reported refractive correction was nil or less than 0.37 diopter. The number in this category is 134. This shows that doctors who sent cases to the hospital were thinking of aniseikonia as a possible cause of eyestrain even when there was no obvious anisometropia. They were right in their assumption in a number of cases, though in the majority it was necessary to exclude aniseikonia as even a possible cause of the eyestrain or other difficulty for which relief was being sought by the individual patient. Over-all image size difference of one per cent or more was found in 20 cases in this group, or approximately 14 per cent. In another 50 cases, the difference in refractive correction was given as 0.5 to 1.0 diopter. Seventeen of them,

TABLE II

OVER-ALL IMAGE SIZE DIFFERENCE IN RELATION TO REFRACTIVE DIFFERENCE IN DIOPTERS IN 265 HOSPITAL PATIENTS

REFRACTIVE DIFFERENCE IN DIOPTERS	NO. OF CASES	PATIENTS WITH OVER-ALL IMAGE SIZE DIFFERENCE					
		0.50—0.75%		1.00—1.75%		2.00% AND OVER	
		NO.	%	NO.	%	NO.	%
No error	32	8	25	4	12	0	0
0.0-0.37	134	39	29	18	13	2	1
0.50-1.00	50	8	16	15	30	2	4
1.12-2.00	22	2	9	7	32	3	14
>2.00	27	0	0	3	11	11	41

or 34 per cent, showed over-all size difference of one per cent or greater. In the next group in which the difference was given as 1.12 or 2.00 diopters, there were 10, or 46 per cent, with the same, probably significant, amount of aniseikonia. And finally, again there was that small but interesting group of patients with large difference in refractive correction, over 2.00 diopters, but binocular vision good enough to make determination of aniseikonia feasible. None showed image size difference of less than one per cent, and in some of them values were found which were larger than those encountered in any other group. The number of patients with high over-all *and* meridional image size difference is also significant for the size of this group and the difference also runs at the highest percentage level. I have discouraged several of these patients in their insistence on binocular vision.

It might be a digression from my topic, but I cannot help interjecting here a few words about the compulsion to binocular vision. I don't mean compulsion in the sense used by Ogle, the compulsion in normals to see single when prisms are introduced or other kinds of stress put on the binocular mechanism. I mean the compulsive neurosis of parents who drag their children from orthoptists to surgeons, and back again, driven by the anxiety and guilt modern parents feel if their children are not "normal" in some respect. Binocular vision, or the lack of it, becomes an obsession with them. And this obsession is nurtured by practitioners, who either don't know better or—I may be forgiven for this inference—have an interest in giving exercises. I am not against eye exercises. With the recent advent of pleoptics, about which many of us heard so much at the recent International Congress, I believe that the treatment of *amblyopia ex anopsia* has reached a new era, and that amblyopia has to be combatted by every possible means. I also believe that with the early surgical intervention now practiced, the stage is set for better results than heretofore in the field of binocular training. Much can be achieved that was impossible decades, even years, ago. But I am still convinced that a teenage girl who is emmetropic, or nearly so, in one eye and myopic in the other—like one of the patients in whom I found aniseikonia of more than 5 per cent—is better off if she is not trained to use both eyes together but is left alone to use one eye for distance, the other for near. It is my conviction that people with compulsive binocular vision *should* be corrected for aniseikonia if it interferes with the efficiency of binocular vision or if it is the cause of eyestrain. Children with defects in binocular vision should be given every opportunity to develop it or improve it. On the other hand, teenagers and, especially, grownups, if they have no compulsive binocular vision, should be left alone.

As long as I am digressing, I might say a few words about another source of parental chagrin, the so-called reading difficulty mentioned earlier. Several patients have been sent to me because of their inability to read, which, by the way, was described for the first time in this country by our great Edward Jackson. He called it "developmental alexia," rejecting the name "congenital word blindness" given to it by earlier European authors. Jackson had already noted that errors of refraction cannot be blamed for it, though in all the cases he found in the literature the patients were hyperopes. Nor can aniseikonia be blamed, at least not generally speaking. I did have a few cases in which reading skills were picked up with remarkable speed after this obstacle to binocular coordination was found and removed. But some of these children will improve in reading as soon as something, anything, is being done for them, breaking what

seems to be a vicious circle. In the majority of instances, I must confess, results of eye consultations in cases of retarded reading faculty are disappointing—disappointing to parents who expect something tangible, something physical which they can blame for an inability for which there is actually no pathologic background. Reading difficulties in the Anglo-Saxon *Kulturkreis* are caused by several factors. One is the inability, or reluctance, to shed superficial manifestations of the sacred past: beefeaters wear uniforms and justices wigs which are outmoded, expensive and uncomfortable. A similar spirit keeps spelling unnecessarily complex and archaic. For no good reason but the historical, the vowel in *speak* and *speech* sounds the same but is spelled differently, while in *break* and *breach* it is spoken differently. (It would be redundant to mention *bread* as a further example of the same letter combination pronounced in a still different manner.) Children are logical beings: they crave rule and order. The haphazard complexities of the grownup's world are strange to them and frustrating. The obvious inconsistencies of spelling make the learning of reading and writing in English unnecessarily difficult. A second important factor is the egalitarian character of education. Our schools don't know what to do with the gifted child, and our teachers have no time to devote to those less capable than the so-called average. However, the main offender is the preposterous idea of teaching sight reading instead of phonics at the elementary level. This really puts the cart before the horse. A young mother, all in a panic, brought her lovely son to me because she was told that her child suffered from strephosymbolia, that the pictures in her child's brain were reversed. He mixed up *was* and *saw* in the reading test. The teacher taught him to read the following "word picture"

W A S

as *was*, and the following word picture

S A W

as *saw*, but she was a proponent of sight-reading and forgot to teach that one reads letters within words from left to right. This little boy was a genius. In spite of his teacher he found out that the two unrelated word pictures consisted of the same picture elements, but he had no lead as to the concept of directional perception of these elements, which is the basis of all reading based on alphabets. There is no intrinsic reason why the letter combination *o* on the left and *n* on the right should be read as *on* rather than *no*. I am convinced that reading from left to right has nothing to do with the dominance of the left brain. The Phoenicians, the inventors of our system of writing, wrote from right to left. Some of the old Eastern Mediterranean people wrote *boustrophedon*, one line from left to right, the next from right to left, and so on, alternately. Semitic writing continues to go from right to left. Many of us, like myself, brought up in the traditions of more than one culture read with equal facility in both directions, though we always write with the same hand—with the right if we are right-handers. Scholars familiar with the history of writing generally agree that the final pre-eminence of the "abductor" type of writing (i.e., from left to right with the right hand) was due to technical advances in writing materials and the introduction of the brush and quill as writing utensils. It could have nothing to do with any preponderance of one side of the brain. I was three years old when I simultaneously learned to read Hungarian, German and Hebrew. I suffered no strephosymbolia and read the first two from left to right and the last in reverse order. Dr. Jackson agreed with

Nettleship that the best method of combatting alexia is the teaching of reading at as early an age as is possible. And I don't think he referred to sight-reading. Crisp, in his beautiful paper on reading difficulties, went even further. He threw the whole false concept of so-called reading readiness overboard, and urged mothers to take things into their own hands by teaching their children the alphabet even before they entered school. A child being taught to read and play music must first learn the individual notes and then acquire facility in playing the scales. Grasping the meaning of a piece of music comes later. Temporal order is inherent in reading the music as well as playing it and in grasping its meaning. One does not grasp *no* as a symbol outside of time. We don't read Chinese. One pronounces the *n* as a sound element preceding the sound element *o*, and even in the fastest reading, when we allegedly see whole words or several words simultaneously, the temporal sequence is always present. There is, unfortunately, no time to dwell on this subject any longer.

I have to say a few words about metamorphopsia, a condition in which patients spontaneously report that with one eye they see things smaller than with the other. Metamorphopsia is not aniseikonia. Patients with so-called *retinitis macularis exudativa* have micropsia in their affected eye because edema has separated the retinal elements in the macular region. Usually they also notice a marked distortion of straight contours, obviously because the derangement of cellular architecture occurred in an irregular manner. Since several of these patients were sent to my clinic to be measured for aniseikonia, I had to test them. They only added to the number of patients seen, but they themselves derived no benefit from my examination.

The most impressive cases of aniseikonia are the very rare traumatic cases. I have encountered two in the last two years in which an accident to one cornea, a perforating injury with iris prolapse, fortunately not affecting deeper structures, had caused unilateral oblique corneal astigmatism in an eye which heretofore had been essentially like the other eye. These patients had what one could almost call "acute" aniseikonia. The size difference they revealed in the eikonometer test was practically predictable and commensurate to the optical defect.

As far as acute aniseikonia is concerned, we all know, of course, of cases in which refractive correction was changed and the patient suddenly was heard to complain of slanting desks, of trapezoid newspapers, or of feeling himself a foot or two taller or shorter than usual. These were the cases which Jackson accepted as genuine examples of aniseikonia. He was sure that the mind gets used to these phenomena, but he believed that nothing had to be done about it as long as the refractive correction was proper. He certainly was right in some cases, though surely not in all. Often the mind gets more used to a correction if it is incorrect, so to speak, because the retinal image is not quite so sharp and the long accustomed pattern of suppression less disturbed. The small number of anisometropes in my private practice who are given size lenses testifies to my utmost efforts to straighten out my refraction problems without resorting to this type of correction. Reduction of cylindric lens power in the nondominant eye is the most successful, usually, among these procedures. I often almost indulge in the art of inaccuracy. It *is* an art, I confess. In any event, it is the distillation of experience of many years. Here are two examples. The first, an executive, 49 years old, who until about two years before he was sent to

me had no eye trouble. The first and only pair of glasses he ever was given read:

O.D. ⌒ -.75 D cyl, axis 180
O.S. +1.75 D sph ⌒ -.50 D cyl, axis 178
add + 1.00 D, O.U.

He could never wear these glasses. He saw double. Though at his referring doctor's request I tested him for aniseikonia (and with his own glasses he revealed a lot of it), I suggested the following simple reading correction:

O.D. + 1.00 D sph
O.S. + 2.50 D sph

to the patient's great satisfaction.

The second example, a housewife, 48 years old, wore the following correction given by the referring doctor:

O.D. +1.50 D sph ⌒ -.50 D cyl, axis 30
O.S. -1.25 D sph ⌒ -1.50 D cyl, axis 180
add +1.50 D, O.U.

The left eye was dominant for close work and I ordered:

O.D. = plano
O.S. +.75 D sph ⌒ -1.50 D cyl, axis 180.

The young salesman I noted earlier in this lecture (the aniseikonic clinician's dream case, I might call him) could certainly never be handled in this manner. That was a case which fully fits into Jackson's specifications: that both eyes present "ametropia of low degree distinctly differing in kind and degree." As I already mentioned, this patient had small myopia in one eye, small hyperopia in the other eye, with astigmatism against the rule in the former, astigmatism with the rule in the latter. In his case only accurate correction with full correction of aniseikonia gave relief.

An unusual case of acute aniseikonia which taught me quite a lesson is probably worth mentioning. An elderly woman, wearing the following refractive correction:

O.D. -2.00 D sph ⌒ -2.00 D cyl, axis 90
O.S. -1.50 D sph ⌒ -2.75 D cyl, axis 85

came to see me with a recent macular degeneration which reduced vision to 20/200 in the left eye. She wanted new glasses. There was some small change needed in the right eye. Since I could not improve vision in the left eye, I wanted to make the lenses less expensive and prescribed only a -1.50 D sph for that eye. The patient returned, indignantly complaining that with her new lenses all was lopsided and floors and walls were distorted. Though she had hardly any central vision in one eye, the power of peripheral fusion, emphasized by Burian, made itself manifest. I learned my lesson but lost a patient.

At the end of this lecture, I should mention two, if small, pieces of research to which my clinic at the Manhattan Eye and Ear Hospital contributed. Two groups of patients were found in whom more than usual image size differences can often be detected and their correction seems sometimes desirable, often feasible. In the first group belong the patients who have been successfully operated upon for unilateral retinal detachment. In some of these cases, quite by accident, I discovered that the image in the operated eye was smaller. I should not say *quite* by accident. Dr. David Webster, Surgeon-Director at Manhattan Eye and Ear Hospital, a wise clinician, honored friend and supporter of my work, sent me the first patient, a man of 65, whom he had successfully operated upon for retinal detachment of the right eye and who complained of much distress after the operation. The findings pertaining to this case are summarized in the following lines:

O.D. +1.00 D sph ⌒ -1.50 D cyl, axis 95
= 20/30
O.S. + .75 D sph ⌒ -1.25 D cyl, axis 95
= 20/25

There was a right hyperphoria of 4 prism diopters present. Testing for aniseikonia revealed that the right image was smaller by 2.5 per cent vertically, 5 per cent horizontally. Size correction worn now for several years gave considerable relief.

I have, of course, only limited experience. There are still too few cases of retinal detachment in which both the eye operated upon and the other eye have sufficiently good vision to make these patients fall into the category of people in whom measurements for retinal image size differences are feasible. Even smaller is the number of cases in which, after a successfully conducted test of measuring aniseikonia, I found it advisable to correct this condition. The differences were again usually much larger than those encountered in normal pairs of eyes and the compulsion to binocularity much reduced. To my dismay, I find that even after what is considered a successful detachment operation, still more people consult me for a visual aid than for the restoration of their binocular functions.

In any event, the cases aroused the interest of Dr. Donald Shafer. He and his associate, Dr. Brian Curtin, have sent me twelve of their patients who had been successfully operated upon, for image size determination. Dr. Curtin presented these cases at the meeting of the Pan-American Association of Ophthalmology last year, and his presentation aroused considerable interest. One case, especially worth mentioning, could be tested before and after operation for a small peripheral detachment in the left eye.

L. I. (left eye affected)

O.D. + 2.00 D sph ⌒ -.75 D cyl, axis 5 = 20/20
O.S. + 2.00 D sph ⌒ -.75 D cyl, axis 5 = 20/25

Left image smaller in vertical meridian by 0.5 per cent. Five weeks after vitreous implant and electro-surgery:

O.D. + 2.00 D sph ⌒ -.75 D cyl, axis 5 = 20/20
O.S. -.50 D cyl, axis 135 = 20/20

Left image smaller in horizontal meridian by 2 per cent.

Interesting in this case is the disappearance of hypermetropia. Having only one such case, I cannot even speculate on the possibile influence of vitreous surgery on ocular refraction.

The other piece of research was initiated at the request of Dr. Elizabeth Constantine and Dr. John McLean, who published a remarkable series of cases of unilateral aphakia corrected with contact lens. I tested several of their patients and found image size differences to be between 4 and 10 per cent, as expected from theory. I do not know of a single case in this series in which it was necessary to correct aniseikonia. Dr. Constantine in her lecture emphasized the fact that these patients were tested and found to possess the faculty to binocular single vision. But showing the faculty to binocular vision in office tests specifically conducted to analyze this faculty (I usually call it "instrument stereopsis") does not mean normal compulsive binocular vision under everyday circumstances. I can only refer to a recent important paper by Ogle, Burian and Bannon on this subject. They, too, doubt that patients with unilateral aphakia corrected with contact lens have, in everyday surroundings, normal (which means compulsive) binocular vision. In fact, their computations convinced them that when aniseikonia is greater than 3 per cent, retinal images fall outside each other's Panum's areas everywhere except within an area of 4 degrees around the point of fixation. Aniseikonia of the magnitude usu-

ally present in the case of contact-lens-corrected uniocular aphakia prevents fusion and causes physiologic rivalry and physiologic suppression in the retinal periphery. These mechanisms work constantly in all of us, on all object details which are outside the horopter, making up the usual background and surround into which our foveal vision is planted. There is no reason why these patients should have eyestrain. The person whose uniocular aphakia is corrected with a regular spectacle lens, however, belongs in an entirely different category. In his two eyes similar retinal images fall way beyond the fusion limits and way beyond the rivalry limits. Like a person with sudden paresis of an extraocular muscle, these patients see double. Spectacle correction of monocular aphakia with some 30 per cent image size difference usually causes confusion and serious visual difficulties. But it does not cause the type of eyestrain which aniseikonia does.

I have an interesting patient in my own practice, a woman, bilaterally aphakic, who wears contact lenses in both eyes. She was extremely uncomfortable with these as well as with previously tried spectacle lenses. Her spectacle refraction is:

O.D. + 12.25 D sph ⌒ + 2.00 D cyl, axis 115
O.S. + 13.00 D sph ⌒ + 0.50 D cyl, axis 45

Her contact lenses, made by Obrig in New York, had 8.05 mm. radius in both eyes, and

O.D. +18.62 D
O.S. +19.37 D

power. Over these contact lenses she now wears a pair of spectacle bifocals with

O.D. 1△ base down ⌒ 2% O. A. magnification
O.S. 1△ base out

in the distance portion of the lens and a suitable reading addition. She is a changed person.

And there are many similar cases. Some have been followed now for more than twenty years at the Dartmouth Eye Institute and in the practice of several of its graduates. It has been possible for us to give relief to some and to lessen inefficiency and agony in others. Even if Dr. Jackson doubted that the initial findings were significant and the results relevant, aniseikonia is a clinical entity of importance and is here to stay.

The title of Dr. Jackson's last paper was "Clinical Importance of Aniseikonia." It is now sixteen years since this paper was delivered. Sixteen years is quite a time to gather experience. There is now sufficient accumulated evidence that the correction of aniseikonia, in suitable cases, adds to the value of refractive correction. The "Clinical Importance of Aniseikonia" is established without any doubt.

THE ELECTRORETINOGRAM IN HEREDITARY VISUAL CELL DEGENERATION

A. D. Ruedemann, Jr., M.D.
DETROIT, MICHIGAN

The modern ophthalmologist, with all his drugs and instrumentation, is stymied by hereditary disease. One form of ocular hereditary disease which has been particularly baffling to the physician is retinitis pigmentosa.

Sorsby[13] classifies this disease among the abiotrophies, intimating that the disease makes itself known after normal visual cell function has been achieved. There are many who would argue with this stand, feeling that the individual with the disease has abnormal function from birth.

In his book "Genetics in Ophthalmology," Sorsby mentions six possible modes of inheritance for retinitis pigmentosa: (1) recessive, (2) dominant, (3) sex-linked recessive, (4) intermediate sex-linked, (5) dominant partial sex-linked, (6) recessive partial sex-linked. The first four are the most generally accepted types. The clinician also must evaluate variation in age of onset in various families, not to mention variability in subjective visual signs (vision, visual fields) and objective findings (ophthalmoscopy). These may vary so much as to make the diagnosis difficult in some cases.

From the Kresge Eye Institute, Detroit.
Presented at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.
Acknowledgment is made of the assistance of Jeanette Shappell and Molly Morrison, technicians, and of Anita Baxter, artist.

In a previous paper[11] we established our criteria for establishing the diagnosis of retinitis pigmentosa. Suffice it to say that in any individual with night blindness accompanied by field changes and typical pigmentation the diagnosis is established. In most of the cases in the original series the family history held no indication of ocular disease of any kind.

Previous publications[2,3,6,7,10] have pointed out certain electroretinographic findings which have not been borne out by our cases. Most authors have claimed that the electric potential of the retina is extinct in true retinitis pigmentosa. Recently Skeehan, Passmore and Armington[12] described a single case with a definite though abnormal electroretinogram. Armington and Schwab[1] described one case. Goodman, in an abstract for the XVIII International Congress of Ophthalmology, noted that individuals with dominant retinitis pigmentosa had intact photopic electroretinograms and reduced scotopic electroretinograms in the early stages of the disease. The electric potential of the retina was extinct in certain long standing cases.

Our studies, from a series of over 60 cases, may be summarized as follows:

1. In an individual with long standing disease and visual fields reduced to within 5 degrees of fixation the electric potential of the retina was extinct, regardless of stimulus.

2. Retention of peripheral field with a midperipheral, or ring, scotoma was associated with definite but reduced and abnormal electric potential of the retina. These cases with the most marked reduction in all components of the electroretinogram had the smallest peripheral fields. In cases with no peripheral field and only 10 to 20 degrees of field, mainly photopic elements were retained.

3. Both a- and b- waves were reduced in these cases, usually not to equal extent, as illustrated in the electroretinograms obtained in three cases previously published (fig. 1). In some cases the a-wave was more prominent than the b-wave (see F in the figure), whereas in others (see G in the figure) the a-wave appears low in relation to the b-wave. In all these cases, the early (x-) component of the response to red light was relatively well preserved and less reduced than the slow, scotopic component of this response.

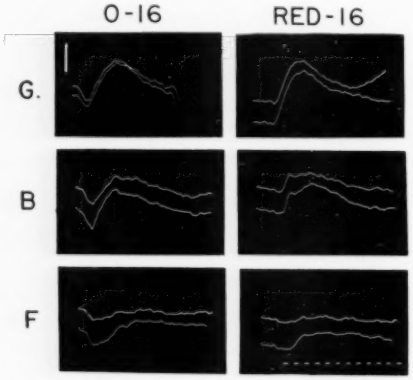

FIG. 1—Vertical line = 100 mv.; horizontal line = 50 msec. Several cases from a previously reported series showing variations in the reduction in the electric potential of the retina in retinitis pigmentosa. (From Ruedemann and Noell.[11])

4. The findings appeared to be the same regardless of causation or hereditary background. Both eyes were involved symmetrically.

The purpose of the present paper is to extend and relate the findings in the previous case studies to whole families in which more than one member has hereditary visual cell disease. We shall also compare two other hereditary visual cell abnormalities with retinitis pigmentosa. Furthermore, the findings in patients will be briefly related to the electrical abnormalities recorded in hereditary visual cell degeneration in mice.

METHOD

The electroretinographic technique used was the same as that previously described.[11] After maximum dilatation with 10 per cent Neosynephrine (this was tested by lack of pupillary response to bright light stimulus), the patient was placed in the examining room and seated in a comfortable chair. The examining method was then explained and the eyes were anesthetized with topical anesthetic (usually 0.2 per cent Dorsacaine). Electrode paste was placed on the forehead and a headband was adjusted, with electrodes to both sides of the forehead for reference, one to the center for ground, and one connection each for right and left corneal electrodes. Modified lacrilens-type corneal lenses with silver button contacts were then placed over each cornea and the electrodes were connected to the headband. At this point the patient was instructed to sit back and relax. Following this, the Grass Photic Stimulator Reflector was advanced to a position one meter from the eyes. (All this time the patient had been in the examining room under conditions of reduced illumination.)

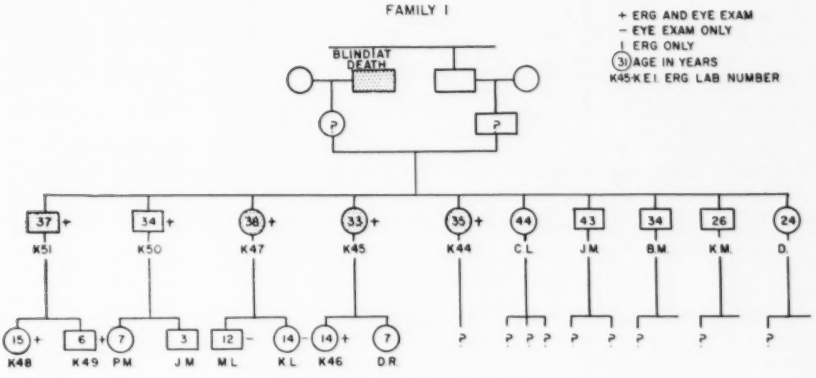

DIAGRAM I—Genogram, Family I

With the patient in the testing position, relaxed and reasonably comfortable, the standard protocol begins. First, the eyes are flashed at short intervals in reduced illumination with minimal intensity stimuli, no filter. Records are taken from a double beam cathode ray oscilloscope by Polaroid Land camera and are recorded from both eyes simultaneously. The stimulus triggers the synchronized cathode ray beam, which is adjusted routinely to a sweep speed of 20 milliseconds per centimeter. Amplification: 200 microvolts give 1.5 inches cathode ray deflection at the screen. Amplification is changed during the test with a 2:1 ratio as needed; calibration is repeated for each patient. The time constant of the system is routinely 300 milliseconds (msec.).

Lights are then turned off. After five minutes of dark adaptation the test is resumed. Flashes are presented at stimulus settings 1, 4, 16 with no filter interposed. Responses are recorded singly and superimposed with a reasonable interval between flashes. No more than five flashes are presented at each intensity. Flashes at intensity 16 are one minute apart. (Maximum intensity, according to specification: at 10 inches, 1,500,000 candlepower, during 10 microseconds.) After a one minute delay, a standard Grass red filter (wave length, 600 to 650 millicron) is interposed and the eyes are stimulated to red. The red filter is then removed, and the eyes are stimulated to flicker at 20 and 50 per second (intensity 4). In each case the same oscilloscope, amplifier and stimulator settings are utilized.

The amplitudes of a-, b- and x- waves from their start to their peak were measured for each record, as well as a-wave latency and x- and b-wave peak times. In this paper we shall report chiefly on the potentials in response to the maximal stimulus (intensity 16) with and without red filter.

FAMILIAL RETINITIS PIGMENTOSA

Family I (diagram I, table I, fig. 2)

The ten siblings in this family descended from consanguinous parents had advanced retinitis pigmentosa with reduced vision and constricted fields. The maternal father was blind at death. The electric potential of the retinas of the five siblings tested was extinct (K44, K45, K47, K50, K51). None of the other

TABLE I
SUMMARY OF FINDINGS: FAMILY I

NAME	PRESENT AGE	VISION	FUNDUS	FIELDS	AGE AT ONSET	DO-16 MICROVOLTS			D-RED 16 MICROVOLTS	
						A-WAVE	B-WAVE		X-WAVE	B-WAVE
							SHARP	SMOOTH		
K51	37	R 20/50+1 L 20/100	Typical R.P.	Constricted	27	extinct	extinct	extinct	extinct	extinct
K48	16	R 20/20 L 20/20	Normal	Not tested	No Sx.	R 200 L 200	R— L—	R 455 L 466	R 33 L 28	R 44 L 33
K49	6	R 20/20 L 20/20	Normal	Not tested	No Sx.	L 233	L—	L 566	L 144	L 250
K50	34	R 20/100 L 20/100	Typical R.P.	Constricted	23	extinct	extinct	extinct	extinct	extinct
K47	38	R LP L LP	Typical R.P.	Vision too poor	28	extinct	extinct	extinct	extinct	extinct
M.L.	12	R 20/40+ L 20/40+	Normal	Not tested	No Sx.	Not tested			Not tested	
K.L.	14	R 20/30+ L 20/30+	Normal	Not tested	No Sx.	Not tested			Not tested	
K44	35	R 20/100 L 20/100	Typical R.P.	Vision too poor	20's	extinct	extinct	extinct	extinct	extinct
K45	33	R LP L LP	Typical R.P.	Vision too poor	20's	extinct	extinct	extinct	extinct	extinct
K46	16	R 20/20 L 20/20	Normal	Not tested	No Sx.	R 216	R 581	R 606	R 158	R 116

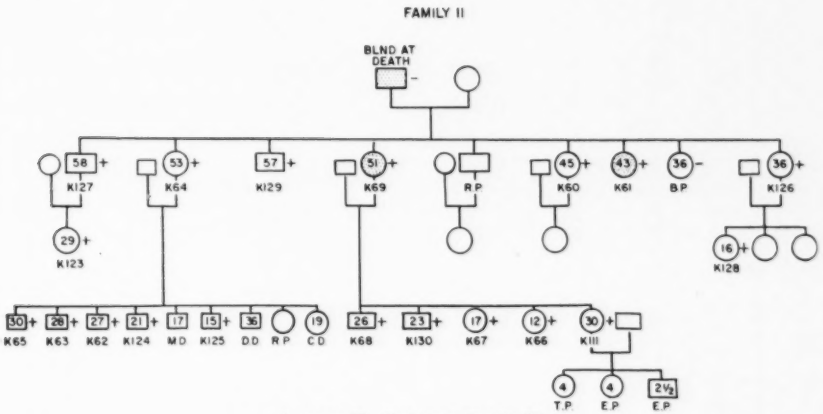

DIAGRAM II—Genogram, Family II

siblings were available for testing. Visual symptoms in each case had not developed until the patient was between 20 and 30 years old. All siblings tested claimed that they had had adequate night vision prior to onset of symptoms.

Two children of K51 were tested both clinically and by electroretinograms and were found to be normal. One child of K45 was tested and found normal. Two children of K47 were examined clinically and there was no evidence of disease.

Family II (diagram II, table II, figs. 3 to 5)

From this large family, two sisters (K61, K69) out of nine siblings have typical retinitis pigmentosa. Clinical records established the diagnosis in the father without question. In all three cases, the disease had its apparent onset around age 40.

K61, age 43, has noted visual symptoms for only two or three years. Visual fields show the typical ring scotoma (fig. 3). All clinical findings, including dark adaptation measurements, are typical of retinitis pigmentosa. The electric potential of the retina of this patient (figs. 3 and 5) is practically as reduced as that of her elder sister, even though her symptoms are of shorter duration.

K69, age 51, has noted visual symptoms for five to ten years. Clinical examination in October 1956 established the

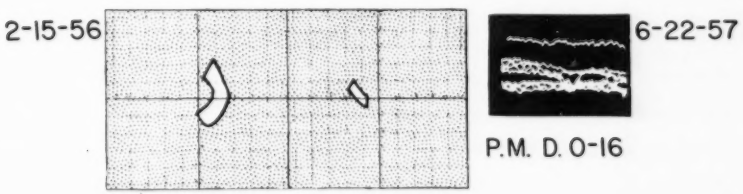

FIG. 2—Typical visual field and electroretinogram of the afflicted members of this family.

TABLE II
SUMMARY OF FINDINGS: FAMILY II

NAME	PRESENT AGE	VISION	FUNDUS	FIELDS	AGE AT ONSET	DO-16* MICROVOLTS			D-RED 16† MICROVOLTS	
						A-WAVE	B-WAVE SHARP	B-WAVE SMOOTH	X-WAVE	B-WAVE
G.P.	dec.	LP only	Typical R.P.	Not obtainable	40's	Not tested			Not tested	
K127	58	R 20/20 L 20/20	granular pig. choroid	Not tested	No Sx.	R 178 L 178	R 411 L 444	R 366 L 389	R 89 L 111	R 78 L 133
K123	29	R 20/20 L 20/20	granular pig. choroid	Full but one hazy spot in periphery LE	Spots	R 178 L 167	R 366 L 344	R 411 L 344	R 67 L 61	R 189 L —
K64	53	R 20/50 L 20/40—2	granular pig. choroid	Full	No Sx.	R 200 L 155	R 400 L 377	R 433 L 400	R 44.4 L 44.4	R 144 L 88
D.D.	36	—	—	—	No Sx.	Not tested			Not tested	
K65	30	R 20/40 L 20/20	granular pig. choroid	Normal	No Sx.	R 266	R 333	R 333		
K63	28	R 20/20 L 20/20	granular pig. choroid	Normal	No Sx.	R 133 L 167	R 311 L 289	R 222 L 272		
K62	27	R 20/20 L 20/20	granular pig. choroid	Normal	No Sx.	R 200 L 222	R 355 L 377	R 366 L 389	R 33 L 44	R 33 L 33
K124	21		granular pig. choroid		No Sx.	R 200 L 200	R 455 L 455	R 433 L 444	R 78 L 89	L 78
M.D.	17	—	—	—		Not tested			Not tested	
K125	15	Normal	granular pig. choroid	Not tested	No Sx.	R 222 L 244	R 555 L 555	R 577 L 555	R 67 L 44	R 155 L —
P.D.		—	—	—	—	Not tested			Not tested	
C.D.	19	—	—	—	—	Not tested			Not tested	
K129	57	R 20/20 cc L 20/20 cc	granular pig. choroid	Full C and P	No Sx.	R 222 L 244	R 455 L 466	R 400 L 455	R 67 L 89	R 111 L 155

TABLE II — *Continued*

NAME	PRESENT AGE	VISION	FUNDUS	FIELDS	AGE AT ONSET	DO-16* MICROVOLTS			D-RED 16† MICROVOLTS	
						A-WAVE	B-WAVE SHARP	B-WAVE SMOOTH	X-WAVE	B-WAVE
K69	50	R 20/30 L 20/30	Typical R.P.	Reduced	40-45 years	R 56 L 56	R — L —	R 139 L 133	R 44 L 44	R 33 L 11
K68	26	Normal	granular pig. choroid	Normal	No Sx.	R 200 L 178	R 466 L 488	R 466 L 477	R 78 L 44	R 111 L —
K130	23	R 20/50 L 20/20	granular pig. choroid	Not tested	No Sx.	R 300 L 255	R 444 L 511	R 455 L 533	R 33 L 22	R 56 L 89
K111	30	R 20/20 L CF 5′	granular pig. choroid	Not tested	No Sx.	R 289 L 244	R 622 L 577	R 644 L 577	R 111 L 78	R 322 L 233
K67	17	R 20/20 L 20/20	granular pig. choroid	Normal	No Sx.	R 244 L 266	R 488 L 533	R 466 L 511	R 67 L 67	R 200 L 167
K66	12	Normal	granular pig. choroid	Not tested	No Sx.	R 155 L 244	R 389 L 422	R 400 L 444	R 44 L 44	R 144 L 67
R.P.	46	—	—	—	—	Not tested			Not tested	
K60	45	R 20/20 L 20/20	Normal	Normal	No Sx.	R — L 278	R — L 483	R — L 466	R 167 L 144	R 233 L 200
J.A.		—	—	—	—	Not tested			Not tested	
M.A.	6	—	—	—	—	Not tested			Not tested	
K61	43	R 20/20 L 20/30—	Typical R.P.	Reduced	40	R 44 L 44	R — L —	R 161 L 167	R 56 L 56	R 0 L 0
B.P.	36	R 20/30+3 L 20/30+3	Normal	Normal	No Sx.	Not tested			Not tested	
K126	36	R 20/15 L 20/15	Normal	Normal	No Sx.	R 155 L 155	R 466 L 455	R 444 L 433	R 89 L 89	R 111 L 89
K128	16	R 20/20 L 20/20	granular pig. choroid	Normal	No Sx.	R 255 L 200	R 500 L —	R 455 L —	R 67 L 67	R 56 L 44

*Normal range for DO-16: a-wave, 150-300; b-wave sharp, 350-550; b-wave smooth, 350-650.

†Normal range for D-Red 16: x-wave, 40-120; b-wave, 60-250.

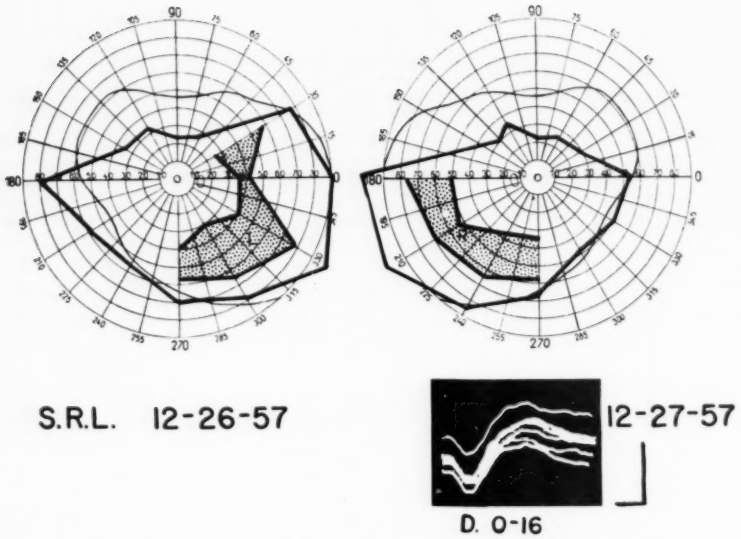

FIG. 3—K61, age 43, symptoms for two or three years.[11] Retinitis pigmentosa.

diagnosis, and re-evaluation of the visual fields in February 1958 confirmed the progress of the disease. The electric potential of the retina has been tested on repeated occasions during the past year; while not extinct, it is certainly reduced and abnormal. From the records illustrated (figs. 4 and 5), all electroretinographic components may be noted, though at much reduced amplitudes. The b-wave is more depressed than a-, x- and flicker waves. Noteworthy is the flicker response to intensity 4. Both sisters have rather well preserved flicker response. The maximal amplitude to flicker is about one-third to one-fourth that of the total response provoked by a single maximal stimulus in the dark. In our normal cases this ratio is 1:6. The response to red is also abnormal in that the usual slow rising b-wave is not evident, but the x-wave or photopic component is well preserved.

In summary, the two sisters have reduced b-wave to maximal intensity white and red with maintenance of rather good flicker. The scotopic components would thus appear more seriously affected than the photopic components. Central visual acuity is within normal limits for both sisters.

K69 has five children (K66, K67, K68, K111, K130) ranging in age from 12 to 30 years. All five have been examined clinically and by electroretinography. None of them have clinical evidence of the disease, but one son and one daughter (K66, K130) have b-waves to red or white in the low normal range.

To date we have examined five of the brothers and sisters (K127, K64, K129, K60, K126) of K61 and K69. The one brother not examined claims to be normal, and the one sister not examined by electroretinography had had a complete eye examination at an earlier date and

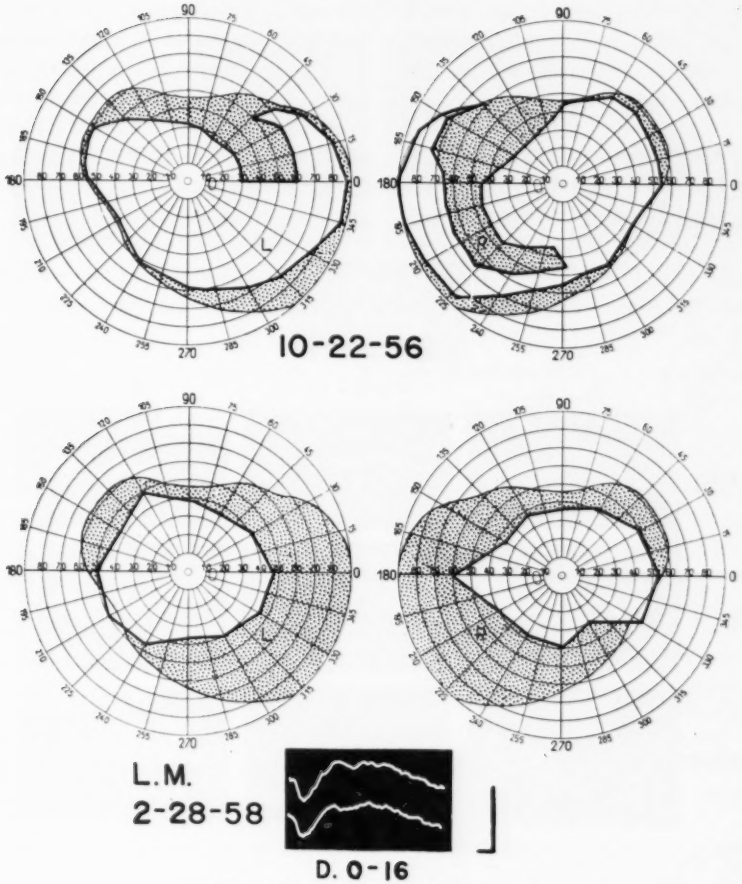

FIG. 4—K69, age 51, retinitis pigmentosa.[11]

was found normal. She and K126 are twin sisters, 36 years old. The electroretinogram of K126 was normal except for relatively low amplitudes to DO-16 and D-Red 16. The 16 year old daughter of K126 (K128) was also examined and found to have relatively low b-waves to red stimulus. The clinical examinations of both mother and daughter revealed no evidence of retinitis pigmentosa except for a characteristic pigment clumping in the choroid which was noted in the entire family.

K60, a 45 year old sister, was found within normal limits both clinically and electroretinographically. A brother (K129), 57 years old, was also within normal limits. Another brother (K127), 58 years old, had scotopic electroretinographic amplitudes at the lower limits

of normal. His daughter (K123), 29 years old, had even lower scotopic amplitudes. Another sister (K64), 54 years old, had a relatively low b-component to DO-16 and D-Red 16 (fig. 5). She has nine children, and five (K62, K63, K65, K124, K125) have been examined to date, both clinically and electroretinographically. The electroretinogram for one son (K63), aged 28 years, shows DO-16. The b-wave amplitude for DO-16 is reduced and the fast cornea positive component is more prominent than is normal under our testing conditions. This electroretinogram has a certain similarity with that noted in our cases of congenital night blindness to be described below. Except for the youngest brother tested (K125), all other siblings tested had low normal b-wave amplitude to DO-16 or D-Red 16.

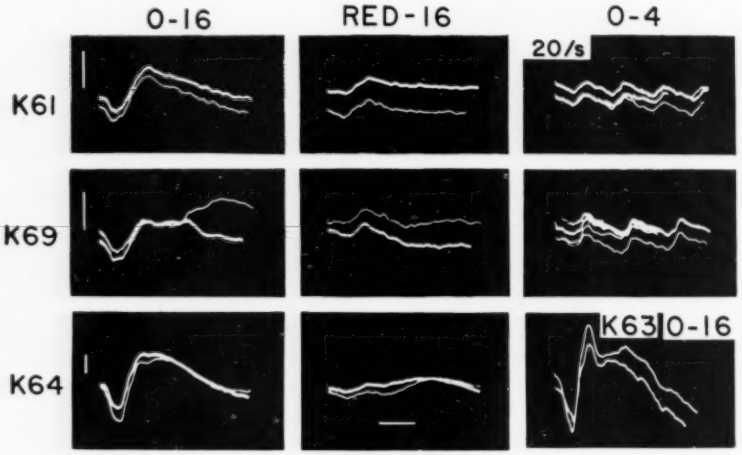

FIG. 5—Vertical line = 100 mv.; horizontal line = 50 msec. K61 and K69 are the involved sisters. K64 is a third sister who did not have clinical symptoms or signs and has slightly reduced b-wave amplitude. K63 is one of her sons who has an abnormal b-wave.

To date we have examined nineteen members of this family. The grandfather had clinically proved retinitis pigmentosa. Two of his nine children have the same disease. There is certainly marked reduction in scotopic components of the electroretinogram. The photopic x-wave response to DO-16 and D-Red 16 is about the only portion of the positive corneal potential remaining.

Of the five remaining siblings examined, K127 and K64 have low normal b-wave amplitudes, but their ages are 58 and 53 years respectively—well past the age of onset in this family. Neither one has symptoms of retinitis pigmentosa. One of the twins (K126) certainly is at the lower limits of normal, but she has no symptoms of retinitis pigmentosa either. Therefore, of the seven examined, two have definite disease, two or three are low normals, and two are normal.

In the third generation, K123, K65, K63, K62, K66, K128 have low normal

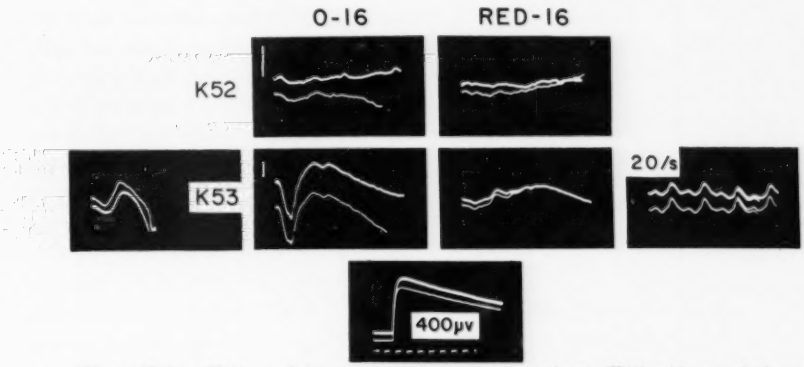

FIG. 6—Mother (K52), aged 58, with retinitis pigmentosa, and son (K53) with normal electroretinogram and no clinical evidence of the disease.

response to DO-16. K123, K62, K124, K68, K130, K66, K128 have low normal response to D-Red 16. *K123, K62, K128* may be said to be in the low normal range to both stimuli.

Since all the requirements of a dominant disease are not fulfilled (e.g., no actual cases in the third generation, and one half of the siblings not involved), one can only say that we are dealing with a hereditary disease which has been proved in a father and two of his nine children. Several of their siblings are in the low normal range, though they are past the usual age of onset of the disease. Twelve members of the third generation have been examined: seven have low normal response to either DO-16 or D-Red 16, and three of these are in the low normal range to both. This would appear to represent clutching at straws except that not one of these patients is over 30 years old, and the members of this family usually do not develop symptoms of retinitis pigmentosa until they are at least 40. Only time can prove the merits of these studies. The value of such a *proof of protoplasm* need not be discussed.

Family III (diagram III, table III, fig. 6)

This family was interesting in that our patient K52 had advanced retinitis pigmentosa and a sister had received a diagnosis of retinitis pigmentosa. The

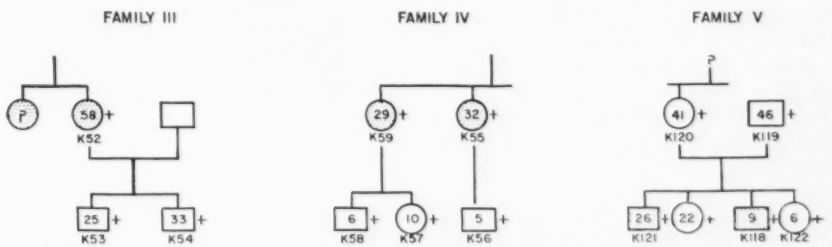

DIAGRAM III—Genogram, Families III, IV and V

TABLE III
SUMMARY OF FINDINGS: FAMILY III

NAME	PRESENT AGE	VISION	FUNDUS	FIELDS	AGE AT ONSET	DO-16* MICROVOLTS			D-RED 16† MICROVOLTS	
						A-WAVE	B-WAVE		X-WAVE	B-WAVE
							SHARP	SMOOTH		
K52	58	R 20/30—1 L 20/30—1	Typical R.P.	<5°	40's	R 22 L 33	R — L —	R 33 L 33	R — L —	R 11 L 11
K54	33	R 20/20 L 20/20	Normal	Not tested	No Sx.	R 344 L 377	R 677 L 644	R 633 L 600	R 78 L 67	R 233 L 266
K53	25	R 20/20 L 20/20	Normal	Not tested	No Sx.	R 289 L 311	R 422 L 444	R 422 L 433	R 111 L 111	R 189 L 144

*Normal range for DO-16: a-wave, 150-300; b-wave sharp, 350-550; b-wave smooth, 400-650.
†Normal range for D-Red 16: x-wave, 40-120; b-wave, 60-250.

case of K52 has been followed since October 1955. In that time vision and fields have remained constant. (VRE 20/30-2, VLE 20/30-2; VF 5° centrally.) Her electroretinogram made on December 19, 1957, is essentially the same as that made on July 29, 1958. It is markedly reduced, with the only measurable component a low and slow a-wave. The electroretinograms and clinical evaluation of two sons of K52 indicate a low normal for K53, age 25 years, and a normal range for his brother (K54), age 33 years. Again only time will prove the value of such measurements.

ALLIED FAMILIAL VISUAL CELL DISEASES

For the purpose of comparison, two families will be discussed. The first family, family IV, has familial macular pigmentary degeneration. The second, family V, has congenital stationary night blindness.

FIG. 7—Sisters aged 32 years (K55) and 29 years (K59) with macular disease. Vision in both eyes 20/200. They are compared to K111, one of Family II who had no ocular disease. Vertical line = 100 mv.; horizontal line = 50 msec. Note the abnormally reduced a- and b-waves, the increased latency of the a-wave to DO-16. The response to DRed 16 is singular in that the x- response is absent and the b- is markedly reduced.

TABLE IV

SUMMARY OF FINDINGS: FAMILY IV

NAME	PRESENT AGE	VISION	FUNDUS	FIELDS	AGE AT ONSET	DO-16* MICROVOLTS			D-RED 16† MICROVOLTS	
						A-WAVE	B-WAVE		X-WAVE	B-WAVE
							SHARP	SMOOTH		
K55	32	R 18/200 L 18/200	central and periph. pig. degen.	large central scotoma	10	R 78 L 78	R — L —	R 239 L 239	R — L —	R 22 L 22
K59	29	R 20/200 L 20/200	central and periph. pig. degen.	large central scotoma	7	R 78 L 89	R 305 L 294	R 289 L 278	R 44 L 44	R 44 L 44

*Normal range for DO-16: a-wave, 150-300; b-wave sharp, 350-550; b-wave smooth, 400-650.
†Normal range for D-Red 16: x-wave, 40-120; b-wave, 60-250

Family IV (diagram III, table IV, fig. 7)

Two sisters, aged 29 and 32 years, developed a slowly progressing failure of vision following a severe "flu" associated with high temperatures. Onset was noted when the sisters were 7 and 10 years old, respectively. Vision was reduced to 20/200 in both eyes. Ophthalmoscopy revealed macular scarring and pigmentation surrounded by diffuse central pigmentary degeneration and mid-peripheral pigmentary changes in both eyes. The lesions noted may be similar to Stargardt's disease. Visual fields revealed large central scotomas; peripheral fields appear within normal limits (not accurate, poor fixation). Both sisters can determine gross color and prefer reduced illumination for best visual acuity.

The electroretinograms of the sisters are similar. The electric potentials in the retinas of each are reduced. Maximum b-wave amplitude is reduced below normal range. K59, b-wave amplitude=300 uV; K55, b-wave amplitude=250 uV. Actually, the most marked effect is noted in the response to red (intensity 16). The x-wave is very small, and there may be increased latency. The reduction in the x-wave response in these two cases would be consistent with a defect in cone function, since the x-wave is notably missing in the totally colorblind individual. The differentiation of this disease on a clinical basis from so-called pigmentary degeneration would be rather difficult except for the preferential involvement of the x-wave in red response. The reduction in the other components may indicate some involvement of scotopic function. In these cases, however, maximum scotopic effect was minimized by the fact that the pupils did not dilate well with mydriatics.*

*We have noted significant reduction in electroretinographic amplitudes if the pupils are not widely dilated.

TABLE V

SUMMARY OF FINDINGS: FAMILY V

NAME	PRESENT AGE	VISION	FUNDUS	FIELDS	AGE AT ONSET	DO-16* MICROVOLTS			D-RED 16† MICROVOLTS	
						A-WAVE	B-WAVE SHARP	B-WAVE SMOOTH	X WAVE	B-WAVE
K119	46	within normal limits	Normal	Not tested	No Sx.	R 253 L 263	R 421 L 421	R 400 L 421	R 42 L 32	R 189 L 211
K120	41	within normal limits	Normal	Not tested	No Sx.	R 222 L 244	R — L —	R 466 L 477	R 122 L 155	R 100 L 200
K121	22	R 20/20 L 20/20	Normal	Normal	No Sx.	R 289 L 266	R 511 L 555	R 455 L 488	R 56 L 89	R 155 L 244
K122	6	R 20/20 L 20/20	Normal	Not tested	No Sx.	R 200 L 200	R 355 L 311	R 333 L 311	R 89 L 56	R 100 L 111
K118	9	R 20/20 cc L 20/20 cc	Normal	Not tested	Birth	R 221 L 221	R 100 L 95	R 89 L 74	R 53 L 42	R 0 L 0
K132	25	R 20/40+2cc L 20/30+1cc	Normal	Reduced	Birth	R 233 L 244	R 100 L 78	R 78 L 67	R 33 L 22	R 0 L 0

*Normal range for DO-16: a-wave, 150-300; b-wave sharp, 350-550; b-wave smooth, 400-650.

†Normal range for D-Red 16: x-wave, 40-120; b-wave, 60-250

The reduction in scotopic components on the one hand as compared with reduction in photopic elements on the other is interesting. Many members of family II had low normal scotopic components.

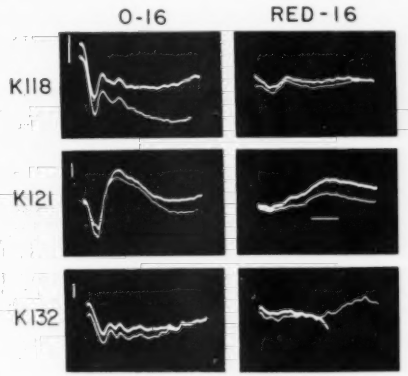

FIG. 8—Two brothers (K118 and K132) with stationary night blindness are compared with their sister (K121). Note the complete lack of scotopic components; e.g., the lack of b-wave to DO and DRed 16.

Family V (diagram III, table V, fig. 8)

This family has two sons with severe nonprogressive night blindness. Neither parent has symptoms of any ocular disease, but the maternal grandfather was blind at death, cause unknown.

The whole family was examined clinically and electroretinographically. Except for the two sons, the results of all ocular examinations were negative. Both sons had circumferential reduction in visual fields to 3/330 white peripherally and 2/1000 white centrally. The electroretinograms of K118 and K132 were similar, yet there is sixteen years difference in their ages. They had the same appearance as that described for familial nyctalopia by various authors.[1,4,5,9] The scotopic elements of the electroretinogram were reduced (fig. 8), with preponderance of the cornea negative (a-wave) components and the fast cornea positive (x-wave) components. The response to red is particularly interesting in that the fast photopic (x-wave) component is clearly evident and the slower b-wave component is absent. The other members of the family had normal electroretinograms.

The electric potential of the retina in familial stationary nyctalopia obviously differs from that recorded in any of our cases of retinitis pigmentosa. This is especially notable in the blunting and increased latency of the a-wave in retinitis pigmentosa, associated with obvious retention of scotopic b- to both white and red (fig. 1) in some cases. Familial stationary nyctalopia has an excellent a-wave with marked reduction in scotopic b- to both white and red. The obvious retention of the rapid corneal positive component (x-wave) is notable in both diseases. The main differences would appear to be the progressive nature of retinitis pigmentosa as opposed to the stationary nature of nyctalopia, and the difference in a-waves. The progressive nature of retinitis pigmentosa is well illustrated by comparing the electroretinogram obtained from G (fig. 1) to the absence of electric response noted in all of family I. Both diseases are hereditary in nature. From our cases the last part of the electric response to disappear seems to be the a-wave and a slight cornea positive component (x-(?)).

The relationship to visual field changes may be hypothesized as follows:

1. In early cases with a ring scotoma and peripheral as well as central retention of field, both scotopic and photopic components of the electroretinogram are present, a-, b-, x- waves are reduced, and latency is increased.

2. When the peripheral field becomes absent, leaving a central field of about 20 degrees, the electroretinogram would ap-

pear to be composed mainly of photopic elements. The a-wave is reduced and slowed, the b-wave is markedly reduced and nearly absent to red light. The x-wave is reduced but present.

3. When the field is reduced to five degrees, the electric potential of the retina is extinct.

DISCUSSION

Visual cell degeneration on a hereditary basis is also well known for several animal species, including horse, dog, rat, and mouse. Generally, degeneration is closely related to visual cell differentiation in these species, and it follows shortly after a certain stage of histologic maturation of the retina has been reached. Parry, Tansley and Thomson[9] studied electroretinographically the progress of degeneration in Red Irish Setters in which the disease was inherited as a mendelian recessive character and resulted in night blindness and lack of electric potential in the retina. The dogs eventually became completely blind. Degeneration of the visual cells began as early as eighteen days after birth; at about fifty days, practically all rod cells had disappeared. In these dogs, the electric potential of the retina developed to a very small degree only and disappeared within ten days after it had made its first appearance.

Noell[8] studied the electroretinographic record during the progress of visual cell degeneration in the C3H mouse. This degeneration is autosomal recessive and is found in each mouse of this inbred strain. The histologic features of this degeneration are practically the same as those described by Sorsby and his colleagues for the Bruckner mouse.[14]

Postnatal visual cell development in the afflicted mice proceeds normally up to the age of 10 days. At this age, the inner layers of the retina appear almost mature and the rods have grown to about one fourth of their adult size. The rods, moreover, are differentiated into inner and outer limbs. Beyond this stage the retina of the C3H mouse does not develop. Pyknosis within the outer nuclear layer, followed by cell lysis, is noted at the age of 11 days. It is first evident in the central region of the retina, where the development of the visual cells proceeds in the mouse faster than peripherally. Once started, degeneration rapidly spreads over the whole retina. At 14 or 15 days the outer nuclear layers are reduced from about eight rows of nuclei to three or four, and to one row at the age of about 20 days. Only the visual cells close to the ora serrata have not deteriorated at this time, but they also vanish within five more days. The visual cell layers all over the retina then have been reduced to one single row of nuclei.

The electric response of the retina in these mice makes its appearance as early as in normal control strains; that is, at the age of 10 days. It increases in amplitude for four to five days, then declines as visual cell death progresses. The electric potential of the retina disappears at about 25 days when the last visual cells at the ora serrata are deteriorating.

The electroretinogram of the C3H mouse, prior to its complete disappearance, has a characteristically abnormal form (fig. 9). Its a-wave is low and slow, and the b-wave develops with longer latency than in the normal strain (DBA mouse). As long as death has not yet involved the majority of the visual cells, reduction in maximal amplitude is slight. It reaches a marked degree only when visual cell death has become overwhelming.

Obviously, this degeneration in the mouse follows a time course different from that in the average patient with retinitis pigmentosa and it may have

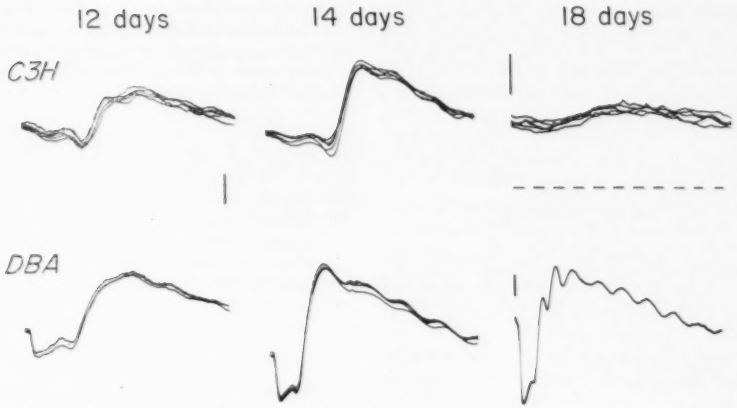

FIG. 9—Electroretinographic record of abnormal (C3H) and normal (DBA) mice at the ages of 12, 14, and 18 days. (Supplied by W. K. Noell, M.D.)

quite different histopathologic features. Nevertheless, it indicates clearly what to look for in the analysis of the electroretinogram in human retinitis pigmentosa. The over-all reduction in amplitudes appears less important than the abnormal relationships in the size of the electroretinographic components. In the mouse, one is overwhelmingly dealing at all degrees of stimulation with responses of the rod system. The changes in the form of the C3H electroretinogram, therefore, denote abnormalities of function within the rod system. Similarly, in human retinitis pigmentosa the electroretinogram contains more information than the relative degree of impairment of scotopic and photopic elements.

In the C3H mouse the electroretinographic abnormalities strongly suggest that outer and inner limbs are functioning abnormally. Similar electroretinographic changes are recorded in rabbits when histologic degeneration of inner and outer limbs is evident after intravenous poisoning by sodium iodate. The electroretinographic changes in the mouse, therefore, appear to reveal that abnormal outer and inner limbs have developed during the differentiation of the visual cells to a functional state. This faulty development is considered to be the first manifestation of an inherited deficiency of the visual cell which eventually leads to its somatic death.

CONCLUSION

Our study of families with retinitis pigmentosa and allied familial diseases gives some indication that a mild deficiency in visual cell function may exist prior to manifestations of the disease or without such manifestations ever developing. To further our knowledge of these diseases, repeated electroretinographic examination of afflicted families probably provides hope because it gives the best chance for the study of the most important, early stages of the disease. Since retinitis pigmentosa, even with regard to abnormalities in the electric potential of the retina, occurs in various forms, it will be necessary to extend such a study over several families

with different types of the disease. This may appear to be a very laborious task, but it seems the best procedure if further progress in the understanding of the disease is to be achieved.

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DISCUSSION

JERRY H. JACOBSON, M.D., New York: I should like to offer my congratulations to Dr. Ruedemann upon his excellent presentation, especially upon the painstaking manner in which the geneologies have been defined. I am going to discuss, if I may, two aspects of the paper.

First, I wish to comment on the use of electroretinography in the diagnosis of retinitis pigmentosa. "Always" and "never" are particularly dangerous words in medicine, I believe, since Nature seems to be too flexible to allow them. Because of this, stating that the electric potential of the retina is "always" extinguished in retinitis pigmentosa has disturbed me for some time. Of the 200 odd patients with classical hereditary retinitis pigmentosa whom I have examined by electroretinography, with but two exceptions, all have had extremely minimal (that is, less than one-tenth the normal amplitude) or extinguished electroretinograms. There were two cases, in a single family, of what appeared to be full blown retinitis pigmentosa with only a moderately reduced electroretinogram. Similar cases from other centers and the beautifully documented cases presented here this morning seem to indicate that, indeed, the word "always" must be discarded.

Yet, I must add a voice of caution in regard to one aspect of this paper. The essayist states that he found definite but reduced and abnormal electroretinographic potentials in those cases with retention of peripheral field and ring scotomas, with greatest reduction in the electroretinogram being coincident with smallest fields. The obvious implication of this is that there exists a correlation between progression of disease and the electroretinographic amplitude.

This is not in keeping with the findings of my laboratory and those of others. There are many cases such as the case of a 25 year old woman, of a family with a clear dominant inheritance, with only minimal fundus changes consisting of narrowed vessels and a few areas of sparse pigmentation, ring scotoma in the fields, but markedly diminished adaptation, and no electric potential in the retina. Certainly this case does not, nor do other cases like it in my experience and those in the literature, agree with this implied correlation.

In family IV, the author found reduction of a moderate degree, whereas in this case [slide] which was clinically very similar, we found no response. We considered this a "central retinitis pigmentosa."

As I read this manuscript, the word "extinguished" is in quotation marks, and I want to place it now in verbal ones, because I want to express my opinion that the term "extinguished" is a poor one. I would much prefer the use of "nonrecordable," signifying that we are unable, by our present techniques, to record a potential change. I feel sure that as our instrumentation improves, allowing us to detect potentials of 2 microvolts or less, there will be fewer and fewer "extinguished" records.

From the clinical standpoint, just what does this report mean? Does it mean that the electroretinogram is no longer of use in the diagnosis of retinitis pigmentosa, and therefore is of little use to the clinician? I think not. To say this would mean that we ought to discard the electrocardiogram because it allows us to differentiate between right and left bundle branch block.

I think, instead, that we are now approaching the state of knowledge of this subject which allows us to make the term retinitis pigmentosa, as a definitive diagnosis, almost obsolete. I believe, that as a result of this excellent study and of others like it over a period of years, the natural history of the progression of this group of diseases, including visual acuity, field changes, fundus changes, changes in the electric potential of the retina and dark adaptation, will be delineated. This will result in the subdivision of the gross group of patients into a number of distinct types; and as a result of this classification, we will be able to offer more accurate prognosis and, perhaps one day, therapy of these diseases. In the meantime, I should like to emphasize that in most cases of hereditary retinitis pigmentosa the electroretinogram is at the vanishing point, and that in my opinion, none of these cases is adequately studied without adaptometry and electroretinography.

Until a specific case or group of cases has been followed from a stage of minimal disease and only moderately reduced electroretinogram through the years to a far advanced clinical picture and a nonrecordable electric potential we will not have established that there is, really, progression of the electroretinographic findings to match the progression of the disease.

As a second aspect of this discussion, I should like to discuss what I have called "spectrodifferential" electroretinography, that is, the use of monochromatic spectral components as stimuli. The author notes, and this has also been reported by our laboratory, that in central retinal disease there seems to be a greater depression of the electroretinogram in response to red stimuli than to other portions of the spectrum.

The clinical importance of this finding is enormous. If we were able to determine the state of macular function when the macula is hidden by lenticular or corneal opacity, we would have an extremely valuable prognostic device.

The difficulty which arises in this determination is the fact that the scotopic components of the electroretinogram are much larger and more easily detected than the photopic ones, and a means must be devised to "uncover" the smaller photopic, roughly the cone, function from that of the rods.

The following techniques have been utilized:

1. Restriction of the stimulus, optically, to the macular area. This has not been found of value.

2. The use of monochromatic stimuli. As noted by the author, and as observed in our own studies, this seems to be of value. The physiologic basis for this technique is the known fact that the scotopic mechanism is less sensitive to stimuli at the long, or red, end of the spectrum than is the photopic mechanism. Since macular function is photopic, using these red stimuli makes the response more representative of central than of total retina.

3. The use of flickering stimuli. This also depends upon a known physiologic fact. It is known that the flicker fusion rate of the central area is higher than that of the periphery. If, then, we use a stimulus flickering at a rate higher than that which the peripheral retina can react to (that is, above the scotopic fusion rate), we have eliminated the peripheral response and again have a representation of the central function.

4. Suppression of the scotopic mechanism by light adaptation.

In a group of 40 cases of far advanced macular degeneration we found that the response to red was absent.

In one patient with a unilateral macular hole there was a marked difference in response to red stimuli, but very little in response to white light.

In accordance with a suggestion of Dr. Hermann Burian, we next tried to eliminate the possibility that the reduction was due to intensity rather than spectrum. This was done by using neutral density filters to vary the light so that approximately equal responses to white, green and red stimuli were obtained in a series of normal eyes. Using these stimuli, it was again found that in far advanced macular degeneration the red response was reduced. This has also been true in a case of congenital cone agenesis. In early cases the technique did not meet with success, so recently we have modified it by the addition of a green background light.

[Slide] This was a case of early macular degeneration. The record reveals that a difference between the better and worse eye was apparent only to red stimuli against a green background. The worse eye alone had a demonstrable central scotoma.

A further refinement is shown in this case [slide], in which there is a demonstrable, but slight, difference between the two eyes of a patient with macular chorioretinitis in their response to red light. This difference becomes more apparent when the eyes are individually subjected to a complimentary color background.

In a few cases of peripheral retinal disease there has been a depression of the white and green responses relative to the red. In color blindness this technique is also of value, as seen in this protanopic record [slide], in which there is a diminution in response to red light.

Again, I should like to thank the author for his extremely interesting and valuable contribution.

WERNER K. NOELL, M.D., Buffalo, N. Y.: I think I would please both the Chairman and the audience if I would abstain from discussing the discussion by Dr. Jacobson. Essentially we are in agreement.

It is the main purpose of the Detroit study to make an electroretinographic record of affected families and thus to detect early and subsequent stages of the disease. The utilization of the electroretinogram for the analysis of retinitis pigmentosa is especially justified by the fact that electroretinography is the best means by which we can relate retinal abnormalities in man to those in animals. In the latter, visual cell abnormalities can be studied biochemically and histologically as well.

During the last years we have studied hereditary visual cell degeneration in mice, mainly in the inbred C3H strain. I should like to show you a few pictures of the histologic manifestation of this disease as it occurs in the mouse.

[Slide] This is the retina of a C3H mouse ten days after birth. The outer and inner layers have grown to about a quarter of their normal length. The inner layers are almost mature, but the visual cells are obviously still at an early stage of their differentiation. Visual cell differentiation stops at this point in the C3H mouse, and within a few days the visual cells die and disappear.

[Slide] This is the retina of the C3H mouse at the age of 20 days. All visual cells have disappeared with the exception of a few nuclei at the ora serrata.

Despite this close association of visual cell death with visual cell differentiation, an electroretinogram develops in the C3H mouse as early as in a normal mouse but it soon shows abnormalities in form. The electroretinogram becomes extinct when the last visual cells have disappeared.

The biochemical cell lesion responsible for cell death might be quite different in mouse and man, but in both, the death of the cells arises from a genetic abnormality. In studying both, and in comparing the electroretinographic findings at various stages of the disease, we hope to be able ultimately to reconstruct the disease process as it occurs in man.

INVOLVEMENT OF THE EYES IN PERIARTERITIS NODOSA

FREDERICK C. BLODI, M.D.
IOWA CITY, IOWA
PAUL B. SULLIVAN, M.D.
HANOVER, NEW HAMPSHIRE

THIS is the case of a 64 year old white female who had suffered from cardiovascular hypertension for at least three years. She had responded temporarily to a variety of drugs and was admitted to the hospital only when she became uremic and when the blood pressure began to rise steadily.

Her hospital course was quite erratic. She varied between profound stupor and complete mental lucidity. There were alarming episodes of gastrointestinal bleeding. Blood pressure rose steadily to 200/150. She became comatose and died one month after admission.

The eye examination contributed significantly to the establishment of the correct diagnosis. On admission, vision in the right eye was 20/40 and in the left 20/200. The retinal arterioles were diffusely attenuated. There was a large, greyish, globular detachment of the left retina. Later, discrete retinal hemorrhages appeared in both eyes.

The retinal detachment together with the bizarre clinical course suggested the diagnosis of periarteritis nodosa. Biopsy of the deltoid muscle revealed the florid changes of periarteritis.

Autopsy further confirmed the diagnosis of periarteritis nodosa involving primarily the cerebral and omental arteries. There was hypertrophy of the heart and pronounced nephrosclerosis. The immediate cause of death was an intracerebral hemorrhage.

Histologic sections of both eyes showed marked hypertensive retinopathy with macular edema (fig. 1), papilledema (fig. 2), eosinophilic deposits in the retina (fig. 3), and retinal detachment (fig. 4). A few areas of active nongranulomatous uveitis were found in both eyes without obvious involvement of the choroidal vessels (fig. 5). An active periarteritis was found only around the episcleral and orbital vessels (fig. 6).

We consider this case of importance because, to our knowledge, it is the first eye with retinal detachment in periarteritis nodosa that has come to histologic examination. There has always been a nice controversy[1] whether such detachment is due primarily to periarteritis of the choroidal vessels or is merely a manifestation of nonspecific, hypertensive retinopathy. While there were inflammatory cells in the choroid, the choroidal vessels themselves were normal. We therefore assume that in our case the detachment was due solely to the hypertensive retinopathy.

Dr. Blodi is from the Department of Ophthalmology, State University of Iowa. Dr. Sullivan is from the Department of Ophthalmology, Hitchcock Clinic, Hanover.

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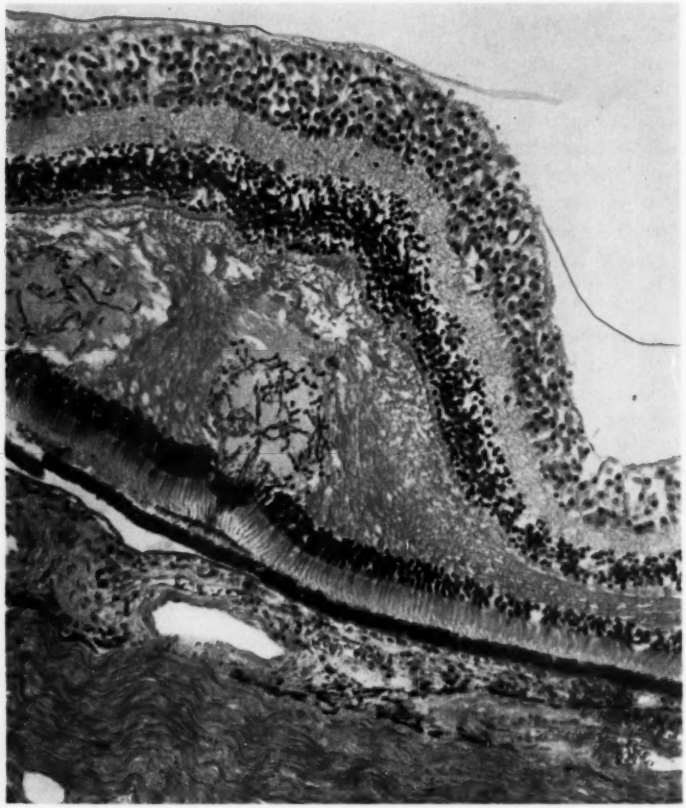

FIG. 1—Edema of the right macula. (×150)

FIG. 2—Papilledema of the right eye. (X33)

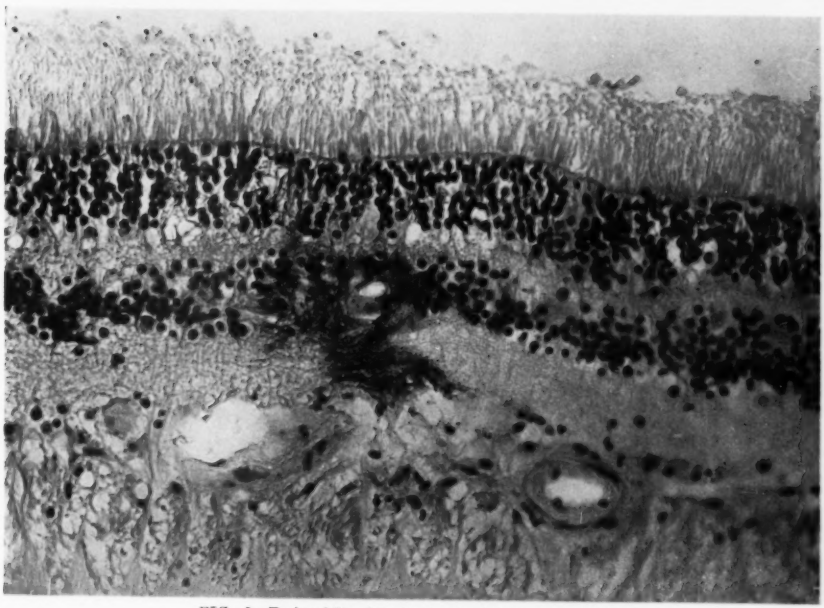

FIG. 3—Eosinophilic deposits in the left retina. (X305)

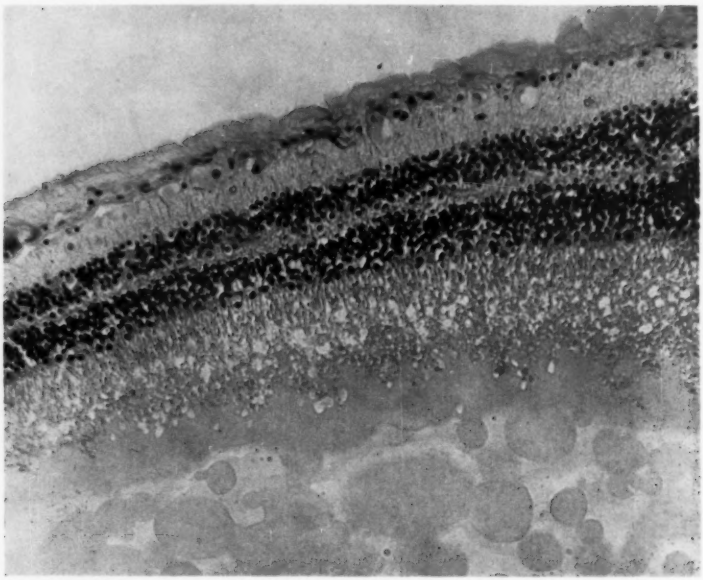

FIG. 4—Retinal detachment in the left eye. (×150)

FIG. 5—Focus of nongranulomatous choroiditis in the right eye. (×305)

FIG. 6—Periarteritis nodosa of an episcleral arteriole in the right eye. (×152)

The choroiditis is of interest. In a clinical study, Woods and Stone[2] found that uveitis and collagen disease did not occur together any more frequently than could be expected by mere chance. Our case shows a disseminated microscopic uveitis. It may be that uveitis occurs more frequently in periarteritis nodosa than the clinician suspects.

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PREOPERATIVE EVALUATION OF THE PATIENT

H. WALTER JONES, JR., M.D.
BOSTON, MASSACHUSETTS

BY INVITATION

OPHTHALMOLOGIC and otolaryngologic surgical procedures are currently highly developed and skillfully carried out. Anesthesia is expertly administered. However, we are confronted by the fact that a significant proportion of our patients suffer from medical diseases, often unrecognized, which predispose them to operative and postoperative mortality and morbidity. This is especially so as the older age groups make up an increasing proportion of our patients and as more radical surgical procedures are undertaken. Our objective must be to make the patient safe for the surgical operation. The purpose of this discussion is to consider the means of achieving this objective.

Who should carry out the preoperative evaluation? In most cases, I believe it should be the surgeon or his resident. He is certainly competent to determine whether a problem exists which indicates calling an internist in consultation. However, I would like to make a special plea that complications be anticipated and that the consultant be called before they arise. There is nothing more frustrating than trying to question a patient who has had a laryngectomy about the nuances of his chest pain.

Assistant in Medicine, Massachusetts General Hospital; Consulting Assistant in Medicine, Massachusetts Eye and Ear Infirmary.

Presented at the *Joint Session* of the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

The preoperative evaluation is begun by talking to the patient, a process commonly known as "taking the history." I like to find out what sort of a person this is so that I can estimate the probability of untoward emotional disturbances. This is the time to anticipate and to plan for the prevention of so-called "black patch delirium."[5] I want to know something about the patient's daily life. If a man can walk up a long hill to the station in the morning without difficulty, or if a housewife can do all her own work and carry the groceries home from the store, I am already reassured about the cardiac status of either as a surgical patient. The work load imposed on the heart by anesthesia and surgical procedures is probably no greater than that of ordinary daily activities.

It is especially important to question the patient about the use of drugs. There is a growing list of commonly used potent therapeutic agents which may lead to surgical complications and even catastrophies if one is not aware of their prior administration. The prolonged use of the adrenocortical steroids, which may masquerade under a variety of trade names, tends to cause adrenal atrophy.[2] A stressful incident such as an operation may then cause the crisis of adrenal insufficiency. Any patient who has been treated for two weeks or more with one of these compounds during the year prior to the surgical operation, or possibly even longer, should be supported

with adrenal steroids before, during, and for a few days after operation. This does not apply to the prior use of cortisone eye drops.

One should also inquire about the use of tranquilizers. Chlorpromazine potentiates the depressant effects of barbiturates and narcotics and may cause serious hypotension and respiratory depression.[4] Shock due to chlorpromazine is resistant to the effect of pressor agents. It has also been reported that the reserpine group of drugs may cause shocklike reactions during anesthesia.[1] These drugs have a prolonged duration of action and it has been recommended that any surgical procedure be delayed two weeks after the last dose. In our experience it has not been practical to do so, and we have not had any trouble.

The patient who is a candidate for surgery may be profoundly disturbed by the abrupt withdrawal, when he enters the hospital, of some substance to which he is addicted. Delirium tremens occurring in an alcoholic patient postoperatively may be fatal. Barbiturates and meprobamate, when taken in large enough quantities, may cause true addiction, with convulsions occurring on cessation of the drug. It is my opinion that drug or alcohol addiction is an absolute contraindication to any surgical procedure except in true emergencies.

While we are talking to the patient about his medications, we should also find out about his allergies. Sensitivity to penicillin is quite common. Some patients, as you know, are allergic to atropine eye drops. There are also a few patients who become psychotic after using scopolamine eye drops. If allergic rhinitis is present, sneezing can usually be controlled with antihistamines.

In taking the patient's history, one should not neglect a review of gastrointestinal symptoms. I have seen hemorrhage occur from a duodenal ulcer following an operation on the retina, and it may be advisable to institute at least a modified ulcer program in patients with a history of ulcer. We have also seen acute cholecystitis flare-up following an operation on the eye. Finally, one should pay special attention in male patients to symptoms indicating prostatic obstruction. It is well to ask if they have difficulty voiding in bed and if there has been acute retention following previous surgical procedures. Acute urinary retention has been the most common serious medical complication in our retina patients. We find it helpful to be forewarned. We allow the patient to stand to void if possible and, if retention does occur, we catheterize the patient before the bladder has become overdistended and lost its tone. Nevertheless, a number of patients have required prostatectomies.

Patients should also be questioned about evidence for hemorrhagic disorders. Many of them claim that they are "bleeders," and a few actually are.

After talking to the patient during the preoperative evaluation, one will make a careful physical examination. Take a good look at the patient. Does he appear older than his chronologic age? Has he lost weight? Does he appear chronically ill or debilitated? A patient of 70 who appears young and vigorous will probably be a good surgical risk. You won't need to ask the laboratory to determine his serum electrolytes or measure his blood volume.

Preoperative evaluation should also include a few simple laboratory procedures. It is impressive how frequently diabetes is discovered by the routine urinalysis. Diabetic acidosis must be corrected before a surgical operation, and the diabetes should be brought under control. Good control is especially important in the prevention of tracheo-esoph-

ageal fistulas in laryngectomy patients. The blood count often discloses significant anemia. Low preoperative hemoglobin levels have recently led to the diagnosis of unsuspected carcinoma of the colon in two retina patients and untreated pernicious anemia in a patient who was to have a laryngectomy. These patients were not only in need of treatment for this dysfunction, but they were poor operative risks until the anemia had been corrected. Hypertensive patients and those with known or suspected renal disease should have a nonprotein nitrogen, or similar, determination. Nitrogen retention is one of the few real contraindications to surgery. Patients with significant hypertension and those with cardiac signs or symptoms should have an electrocardiogram.

Every patient who is to undergo general anesthesia, and also the older cataract patients, should have a preoperative chest roentgenogram. Chest roentgenograms are helpful in evaluating heart size and in detecting pulmonary disease. They are particularly useful in evaluating postoperative complications. A minor change in a postoperative chest film may be the clue to the presence of a pulmonary embolus if we know that the change was not there before. Chest roentgenograms in our experience have paid the extra dividend of revealing carcinoma of the lung in several instances. There is an increased incidence of lung cancer in patients with carcinoma of the larynx, and one should be on the alert to detect them. One patient entered our hospital because of a pain in the ear. The pain went away after a cancer of the lung was removed.

I should like to comment further on a few of the specific problems that may have to be faced in the preoperative evaluation of the patient. I have not stressed cardiovascular problems because, although they are common in our patients, they have not been a frequent source of trouble. Coronary thrombosis, for example, has been a very rare operative complication in our ophthalmologic and otolaryngologic patients. However, careful evaluation and preparation of the patient are important. A history of coronary thrombosis in the preceding three to six months indicates that the operation should be delayed if at all possible. Patients with congestive heart failure should have compensation restored before submitting to any surgical procedure. Minor cardiac arrhythmias such as infrequent premature beats may usually be ignored, but auricular flutter, rapid auricular fibrillation and other major arrhythmias must be corrected. Uncomplicated hypertension is seldom a problem, but patients with extensive cerebrovascular disease are precarious risks. In all of these patients with vascular disease, wide fluctuations in blood pressure must be avoided and a good airway must be provided, not only during operation but in the recovery room as well. Patients with cardiac disease and older patients in general present an increased risk of cardiac arrest, a condition with which all surgeons must be prepared to cope.

Chronic pulmonary disease is common in our patients. A recent special study of a series of patients with detachment of the retina demonstrated emphysema in a significant number.[3] These patients have withstood the effects of anesthesia and surgical operation remarkably well. One should remember that the usual doses of narcotics in an emphysematous patient may depress respiration and that the administration of pure oxygen is also dangerous. Chronic bronchitis with or without emphysema is also common in both ophthalmologic and otolaryngologic patients. Sticky bronchial secretions are often caused, at least in part, by excessive cigarette smoking. We are always apprehensive about putting these patients to bed and are constantly on the

alert for the development of atelectasis. We are also anxious to prevent the eye patients from coughing postoperatively. Often the situation is improved by giving a simple expectorant such as potassium iodide and asking the patient to refrain from smoking for a few days preoperatively. If purulent bronchial secretions are present, a short course of antibiotics may result in marked temporary improvement.

The problem of thromboembolic disease is always with us. This condition may even be present on admission to the hospital in patients who have traveled long distances or who have already been confined to bed. To be constantly alert is the chief prerequisite to preventing catastrophe. For patients with a past history of phlebitis or emboli and those with varicose veins we frequently prescribe elastic stockings to be worn throughout the period of hospitalization and convalescence. We have the impression that this has been helpful. We do not use elastic bandages, which may be worse than useless due to the difficulty of keeping them evenly and firmly applied. If a patient has had a recent episode of phlebitis, we may begin anticoagulant therapy the day after operation.

Disorders of fluid and electrolyte balance should be corrected preoperatively. One should be watchful for the occasional patient with myxedema, hyperthyroidism, or Addison's disease. Anesthesia is dangerous in all of these conditions when untreated. Another complication which is not life threatening but may be very uncomfortable is the tendency of gout to exacerbate following any operative procedure. Appropriate preventive therapy should be instituted.

In conclusion, complicating diseases are common in ophthalmologic and otolaryngologic patients, but it is rare indeed that we need deny the patient the benefit of a proposed operation, and equally rare that a fatal outcome ensues. The purpose of the preoperative evaluation is not only to determine whether the state of the patient is safe for surgical procedures but also to make it so.

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Symposium: Care of the Surgical Patient

FALLACIES IN CHOICE OF ANESTHETIC AGENT

WILLIAM K. HAMILTON, M.D.
IOWA CITY, IOWA
BY INVITATION

THE choice of the proper anesthetic agent for a particular operation has been the subject of much writing and more talking in anesthetic and surgical circles in the past. It is my belief and that of many others that many of these articles and discussions have been based on empirical thought and are not the result of carefully evaluated studies. Continued research and experience have resulted in more information being available; it is appropriate to look more critically at some of the ideas concerning the choice of anesthetic agent. If one assumes that the past ideas concerning choice of anesthetic agent are based on empiricism, it is appropriate to consider why these empiricisms exist. The practice of anesthesia is a relatively new specialty. It has not had time as yet to outgrow ideas and concepts which develop coincident with the lack of complete information. It takes time and experience to have something other than empiricism upon which to base an idea. Furthermore, a large portion of anesthesia in this country is administered by technicians, which probably favors the perpetuation of ideas without subjecting them to continued scrutiny. It should also be noted that the pharmacologic and physiologic information available was and is too meager to supply a completely scientific alternative to vague and incomplete clinical impressions.

In general, the idea of specific choice of anesthesia or anesthetic agent has been based upon two rather broad concepts. The first of these is that certain disease states require or are harmed less by a certain type of anesthetic agent. The second is that there exist certain rather definite contraindications and indications for each particular anesthetic agent. It is my opinion that these concepts are not valid and that benefit from choosing a specific type of anesthesia and a specific anesthetic agent in certain cases has not been borne out by clinical experience. As evidence for my stand on this matter is the fact that many clinics and practicing groups have widely varying choices of anesthetic technique and management with no apparent difference in results. Such would hardly be the case if there were one definitely superior method or one superior agent to be used. Further, it is unlikely that the situations or disease states which have been stated as the basis for specific choice of anesthetic agent would exist in pure culture form often enough to have the situation existing that was present when the original indication or contraindication was described.

Specifically, let us consider some of the indications in these concepts which have been taught in the past:

From the Division of Anesthesiology, Department of Surgery, State University of Iowa College of Medicine.

Presented at the *Joint Session* of the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

1. Coronary artery disease contraindicates use of spinal anesthesia.
2. Heart disease of any type contraindicates use of nitrous oxide.
3. Heart disease is a contraindication to the use of cyclopropane anesthesia.
4. Lung disease or respiratory disease is a contraindication to the use of inhalants for anesthesia, especially the irritating vapors.
5. The presence of diabetes contraindicates the use of ether, and indicates the use of local or regional procedures.
6. Hypertension in a patient is a contraindication to the use of spinal anesthesia.
7. Local anesthesia is the anesthetic choice for the poor-risk patient.

Having listed a few of these concepts, let us consider each of them a bit more closely. First, the common concept that heart disease contraindicates the use of cyclopropane is based upon the belief that arrhythmias are due to the toxic effect of the agent. Recent evidence, however, suggests that these undesirable events are due either to overdose of the agent, which can hardly be blamed on the agent itself, or to hypoventilation or asphyxia occurring concurrently with the administration of the agent. One might look at the cyclopropane question in another way and ask: What better agent for a sick heart could one desire than one which is rapid, not unpleasant, very flexible, offers a minimal excitement phase, and probably offers less depression of myocardial function for a given level of central nervous system depression than other inhalation agents? If cyclopropane is to be denied a cardiac patient, one must consider as alternatives the struggling which often accompanies induction of ether anesthesia and the prolonged depression occasioned by it and nonvolatile drugs. Or its deficiencies can be weighed against the discomfort and apprehension which are contingent with some local and regional anesthetic procedures.

The common occurrence of hypotension with spinal anesthesia has led many internists and anesthetists to advise avoidance of this approach to the cardiac patient. Again, one must weigh the overall depression of general anesthesia and frequent episodes of airway obstruction occurring in the unconscious patient. These may result in an asphyxial episode for the myocardium which is greater in degree and more difficult to treat than the hypotension of spinal anesthesia. Nitrous oxide has been withheld from cardiac patients because of the possibility that hypoxia might occur. Certainly one does not wish to administer low oxygen tension to any patient, and especially not to a patient with heart disease. However, nitrous oxide may be given with oxygen tensions significantly higher than exist in room air by adding supplements of either volatile or nonvolatile drugs. This then becomes a very pleasant nonhypoxic type of anesthesia which can and has been used successfully for countless operations upon patients with all degrees of heart disease.

Respiratory disease has also been used as a basis for indicating and contraindicating certain approaches in the choice of anesthetic agent. It has been taught that the presence of upper respiratory infection and/or the presence of parenchymal lung disease has made it mandatory to avoid so-called irritant gases. There is no evidence that the use of ether anesthesia adversely affects the course of chronic diseases such as emphysema, fibrosis, and tuberculosis. In fact, there is evidence that in patients with lung disease, ether may best protect their ability to respire adequately and thus avoid asphyxia during and af-

ter anesthesia. It appears that ether is rather unique in not being depressant to respiration in the usual dose ranges; it might therefore be considered the agent of choice in these conditions. A frequently heard comment is, "This patient has a cold. Let's use a local or a spinal." It is apparently true that ether increases secretions and general anesthesia suppresses the cough reflex. Theoretically then, these conditions would add to the possibilities of postoperative pulmonary complications due to a spread of infection by a large amount of secretion, and by local obstructive phenomena due to improper coughing. However, evidence or proof of this is completely lacking at the present time. A well conducted inhalation anesthesia should, and can, provide a patient capable of coughing and moving about very soon after the conclusion of the anesthesia. It is not at all unlikely that the injudicious use of preoperative and postoperative sedation may contribute as much to pulmonary complications as the actual anesthetic agent. Therefore, it may be much more important to avoid large amounts of premedicating drugs, to treat the respiratory disease preoperatively to its optimum point, and to minimize the use of drugs after the operation which depress cough and respiration than to make a particular suggestion as to the specific anesthetic agent to be used. The feeling that ether is a specific drug of choice in asthma is based on apparently sound pharmacologic evidence with which I cannot disagree. In my own experience, however, the only severe asthmatic attacks in anesthetized patients that I have seen have occurred *with* ether anesthesia. This limited observation does not alter the prior concepts, but it does open them to question.

The diabetic patient has been the subject of much debate concerning the choice of anesthetic agent. Commonly, it is stated that regional anesthesia should be used and that ether should be avoided because it raises blood sugar levels. Experience has revealed very little, if any, difference in the postoperative courses of these patients due to any anesthetic agent. We now feel that controlled diabetes should not be a factor in our choice of agent. It is desirable, however, to manage whatever agent is used in such a way that intake of food and fluids is interrupted as little as possible. The forcing of regional anesthesia upon an unwilling diabetic patient is not to be recommended. The undesirable sights, sounds, and aromas of the operating room are just as undesirable for them as for nondiabetics, and the diabetic patient has enough inconveniences without adding an undesirable experience which is without proved benefit. Anesthetizing a patient with uncontrolled diabetes with acidosis gives considerable concern, but again no one choice of agent has been shown to be definitely better than any other. For instance, ether has been said to upset the acid base balance more than other agents, but it hinders respiration less. The ability to hyperventilate appears to be very essential in the compensation for fixed acid excess. We might therefore do harm by administering an agent other than ether which would deprive the patient of this compensatory function.

Hypertension has been said to be a contraindication to the use of spinal anesthesia—due again to the high incidence of hypotension. The blood pressure drop is probably undesirable, but widespread use of purposeful hypotensive techniques has made us re-evaluate these ideas. A complete re-evaluation has not yet been completed. Moreover, hypotension does not occur with spinal anesthesia alone. It is common with many other methods, especially in severely ill patients, and perhaps the hypotension occurring with vasodilatation is less undesirable than

hypotension due to myocardial depression, which may occur with the general anesthetic agents.

It is unnecessary, impossible and undesirable to make a complete listing of concepts concerning choice of anesthetic agents. However, I would like to mention one more which is of special importance to this audience. This last example is the concept that local anesthesia offers a great increase in safety to the seriously ill patient. There appears to be a semantic comfort to the surgeon and relatives in the fact that just a little local can be used. This is probably true for very minor procedures, but for other situations one must consider the following facts concerning the use of a local anesthetic.

1. Local anesthesia frequently requires heavier premedication or supplementation during the operative procedure. This may then leave a depressed patient who pays less attention to vital functions than he would have if he had received a general anesthetic.

2. There are serious depressant effects from the absorption of drugs used for local and/or topical anesthesia.

3. Patients frequently are less comfortable under local anesthesia either because of the operative procedure or because of the prolonged immobilization. To avoid this we resort to heavier sedation, which is what we were trying to avoid when we selected local anesthesia in the first place. Certainly the patient's comfort should be thoroughly considered in the choice of anesthetic management.

These comments are not intended as a condemnation of local anesthesia. It is a very desirable method of anesthesia with many advantages. It is not a method offering a great increase in safety to the patient in all situations.

These are but a few examples of conditions for which specific indications and contraindications in the choice of an anesthetic agent have been said to exist. In these few examples, an attempt has been made to show that a considerable doubt exists about the accuracy of any ideas concerning the choice of anesthetic agent. It has been stated that one can quote the Scriptures to support all forms of sin; it may be equally true that one can quote pharmacology texts and the literature concerning anesthesia to support any choice of drugs. Since these drugs do have different pharmacologic and physiologic effects, it can only be said that each agent has advantages and disadvantages and that we have not reached the point at which we can state which of these is undeniably beneficial. It is evident from these remarks that the proper *administration* of drugs is of far greater importance than the proper *selection* of drugs. It is equally evident that considerable doubt and ignorance exist and that further study is required for the proper evaluation of drugs used for anesthesia. We must constantly exercise great care that these investigations and studies are not hampered or limited by empirically based ideas and prejudices.

POSTOPERATIVE SURGICAL CARE

ALTON OCHSNER, M.D.
NEW ORLEANS, LOUISIANA
BY INVITATION

IT is important, I think, for all of us to know something about patients, and I want to congratulate the Academy for wanting to know more about patients generally.

I am extremely fortunate to have been associated with two of your eminent members, Dr. Francis LeJeune and Dr. Mercer Lynch, and I have come in contact with a number of the problems they have faced. Obviously, some of my remarks will cover points already presented by Dr. Jones. Although my part of the discussion is from the surgical standpoint, I was extremely happy to see that you had, as your first speaker, an internist to consider the preoperative evaluation of the patient.

PREOPERATIVE CARE

I think an internist should evaluate all of these patients. It is a rule in our organization that, unless it is a strict emergency, no patient is operated upon who has not been cleared preoperatively by an internist. Internists are better able to evaluate these patients and they are more critical than we are. The general considerations in preoperative care are the following:

1. Discontinuance of smoking
2. Avoidance of catharsis
3. Antibiotics and vitamin C in presence of infection
4. Hydration of the patient
5. Correction of blood volume deficiency and anemia
6. Correction of hypoproteinemia
7. Steroids if previously used
8. Psychic and physical rest

Discontinuance of Smoking

Discontinuance of smoking is one of the first requirements in preoperative care. I think this is extremely important, because we all know that smoking produces a bronchorrhea. Bronchial secretion is, without a doubt, the most direct cause of one serious postoperative complication, namely, atelectasis.

A number of years ago I had a patient who was an extremely heavy smoker and who, in the immediate postoperative period, fell victim to this condition. We did not resort to tracheotomy then. We had considerable difficulty in keeping his bronchi clear; he developed atelectasis and pneumonitis and died. Unless it is an emergency operation, every patient who is a smoker should discontinue smoking for at least a week or ten days prior to operation.

I was very happy to hear Dr. Jones emphasize the importance of a chest

From the Ochsner Clinic.

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roentgenogram. I think every patient who is to be operated upon should have one. As he emphasized, those of you who operate upon patients for neoplasia of the larynx may find that the patient has a concomitant carcinoma of the lung. They are both produced by the same thing—smoking.

Avoidance of Catharsis

It took the surgeons a long time to realize that it was not necessary to have the gastrointestinal tract emptied before a patient is operated upon. Certainly catharsis is to be avoided unless one is operating on the colon, which I assume none of you will be doing.

Antibiotics and Vitamin C

In the presence of infection, it is well known that vitamin C is used up much more rapidly and that the infection is difficult to control. There can be definite interference with healing if there is a vitamin C deficiency. Also, in infection, the appropriate antibiotic should be used.

Hydration of the Patient

If dehydration is a complicating factor, the proper hydration of the patient is essential preoperatively.

Correction of Blood Volume Deficiency and Anemia

If there is a blood volume deficiency and anemia, they should be corrected preoperatively. This is particularly true in patients with neoplasia and those with severe infection. We learned this a number of years ago when Dr. Champ Lyons was on our staff. Dr. Lyons and his associates showed that individuals with carcinoma and/or infection frequently have a blood volume deficiency and anemia, which may not be detected if one relies upon the ordinary blood examination consisting of a red cell count based upon the number of cells per cubic millimeter. The hemoglobin is designated as a per cent value. In a patient with blood volume deficiency, frequently associated with hemoconcentration, there may be a normal red cell count, a normal leukocyte count, and also a normal hemoglobin per cent value, but the total circulating cells and hemoglobin are low. We learned this the hard way.

We knew for a long time that some of our patients with carcinoma of the esophagus, of the lung, and of the stomach appeared anemic; yet their blood counts were normal. If we operated upon them, they went into shock early, and we had difficulty with the healing of their wounds. We also learned that many of these patients were benefited by blood transfusions. We did not know why, for although they looked bad, their blood count was all right. However, after receiving blood, they paradoxically developed anemia; that is, their hemoglobin and blood count dropped. We were not only correcting anemia but were overcoming hemoconcentration, which gave a false picture of the true hematologic status. Following correction of blood volume deficiency, the true hematologic picture can be obtained.

In patients with neoplasia or infection, I would urge that a blood volume determination be done. If there is a deficiency, it should be corrected before operation.

Correction of Hypoproteinemia

If there is hypoproteinemia, which is likely in a patient who has considerable difficulty in swallowing, it must be corrected. It is well known that hypoproteinemia and vitamin C deficiency interfere with wound healing.

Steroids

Dr. Jones has already referred to the necessity of determining whether a patient has received steroids. I should like to emphasize the importance of inquir-

ing whether a patient has had steroids at any time within the past year or year and a half, because if he is operated upon and subjected to the stress of an operation, he is likely to go into shock, which cannot be corrected unless there is substitution therapy.

Psychic and Physical Rest

Finally, it is important for the person to have psychic and physical rest.

CARE DURING OPERATION

During the operation it is important to maintain fluid balance by infusion and to prevent anemia by blood transfusion if the patient is losing blood. In certain instances, hypothermia is indicated, particularly if there is a likelihood of anoxia of the heart muscle or of the brain.

In the consideration of therapy there are general and specific types. The continuation of steroid therapy, if previously used, is extremely important, because the patient requires it as substitution therapy.

Sedation is of importance, but it is equally important that the sedation should not be excessive. I would rather have a patient somewhat restless than to have him knocked out completely. There must be relief of pain, but one must be extremely careful not to use an agent which will oversedate him and interfere with his cough reflex. I feel that it is very important that the patient remain able to cough.

POSTOPERATIVE CARE

One of the postoperative states that requires therapy, but which is probably of less importance to ophthalmologists and otolaryngologists than to other surgeons, is *ileus*. One should always, nevertheless, be on the lookout for it. There are two types. The functional type may occur in any individual who has been anesthetized and any individual who has had trauma to his peritoneum or pleura. The degree of ileus depends upon the length of time and the amount of trauma.

Mechanical obstructions can occur in any patient who has previously had an operation and might have had adhesions.

Dehydration is a factor that concerns all of us and must be prevented and corrected by phleboclysis. In the immediate postoperative period, most patients require fluid and sugar. Very few of your patients will require potassium; a good many of mine would because many of mine have longer periods of functional inactivity of the gastrointestinal tract. It is important that sodium be limited in the first twenty-four hours, because there is sodium retention as a result of stress.

If there is *anemia* which has not been corrected during the operation, it is necessary to give blood postoperatively.

Urinary retention is extremely important postoperatively. Dr. Jones succinctly mentioned that one should determine preoperatively, if possible, whether dysuria is present. If one does not determine it beforehand and if the patient has difficulty urinating, an indwelling catheter should be inserted early. It is far better to employ an indwelling catheter until the patient can void than to allow overdistention of the bladder. The use of a catheter does not predispose to cystitis, but distention and loss of tone of the bladder are favorable factors for its development.

Of the *pneumonopathies,* atelectasis is the most frequent and your patients are subjected to this as much as ours are. Atelectasis must be prevented. Forced deep breathing postoperatively is one way. In our recovery room the nurses

are real tyrants. They make the patient breathe deeply and cough to evacuate any bronchial secretion. If the patient has difficulty in coughing, tracheal suction is valuable; no patient should be allowed to retain bronchial secretions. Turning the patient frequently is also important. At times, if tracheal aspiration with a catheter is not successful, bronchoscopy is necessary; and if secretions continue, tracheostomy is frequently life-saving. It is indicated when one wonders if it is necessary. We do not wait until we are sure, because if we do it is frequently too late.

In the presence of atelectasis, antibiotics are indicated because atelectasis predisposes to the development of pneumonitis. Frequently by the prophylactic use of antibiotics, pneumonia can be prevented.

Aspiration of oral contents can occur during and after operation and must be prevented. Otolaryngologists are aware of such a potential danger; after the completion of nose and throat operations, bronchoscopy is performed. I think this step is very important. One can prevent aspiration by operating with the patient in a head-down position. Modern anesthesiologists use intratracheal tubes with inflatable cuffs and aspiration.

Dr. Jones mentioned that *thrombogenesis* does not occur as frequently among your patients as it does among ours. This is true. I think the reason is that so many of your patients are not kept in bed for long periods of time. I am convinced there are two types of venous thrombosis: one is the garden-variety of thrombophlebitis; the other is phlebothrombosis, which is difficult to detect. The clot in phlebothrombosis is a bland thrombus which is not firmly attached to the vein wall; it is likely to become detached and produce pulmonary embolism. This is a serious matter.

These two types are different in their symptomatology and therapy, and they are certainly different as regards their prognosis. The patient with thrombophlebitis lives to suffer; but the patient with phlebothrombosis is a potential fatality, and one must not waste time, but should take immediate steps to prevent the detachment of the thrombus.

There are two factors responsible for the production of the clot in phlebothrombosis. One is circulatory retardation. It is the one thing for which we can do something. "Phlebostasisogenic" factors should be avoided. Another factor is immobility, such as that imposed upon a patient by bed rest. The patient in that case does not move his lower extremities. Phlebothrombosis usually occurs in the lower extremities. Why? Because the patient in bed constantly moves his hands, putting them to his mouth as in eating, combing his hair, and so on, but his legs are kept absolutely quiet. If there is any distention in the abdomen, it causes pressure on the inferior vena cava and produces stasis. Furthermore, in the person who has been operated on there is a decrease in peripheral circulation. All of these things should be avoided as much as possible. Mobilization of a patient, whether by allowing him to be ambulatory or by encouraging him to contract his muscles against a resistance in bed, is valuable. The principal factor for the movement of blood in the venous system is contraction of the peripheral muscles. We make our patients forcibly ventroflex their feet against a resistance.

Deep breathing is of importance because the negative pressure within the thorax is an important factor for the return flow of venous blood. When we are lying quietly, we are likely not to breathe deeply, particularly if it hurts to breathe. Chilling must be prevented because it decreases peripheral circulation.

Thrombogenesis is a predisposing factor and occurs in individuals subjected

to any type of trauma. I do not think it makes any difference what it is, whether it is an accidental trauma, operative trauma, the result of an operation or the result of an invasion of tissue by a neoplastic disease or by infection, there are changes in the blood constituents which tend to increase the clotting of the blood. This is a protective phenomenon. It is a factor that tends to keep us from bleeding to death. It is a factor found in all traumatized patients, and it is a desirable factor if it does not get out of hand. We have found that dehydration probably favors venous clotting and must be corrected. Administration of alpha-tocopherol and calcium is of value in preventing this thrombogenetic tendency. The original research in this field was done by Dr. Jack Kay at Tulane. It demonstrated that alpha-tocopherol and calcium in vitro decrease the clotting tendency. We have continued to use this drug for the past eight years and believe that it is worth while.

It is a little difficult to evaluate therapy of venous thrombosis, because the incidence varies periodically. It appears to occur in cycles. Why, we do not know. About four years ago we tried a clinical experiment which is open to many criticisms. At the Charity Hospital we discontinued the use of alpha-tocopherol and calcium as a routine postoperative measure, but its use was continued at the Foundation Hospital. The staffs were about the same; the period of time was the same; the only difference was that the operations at the Foundation Hospital, generally, were much more extensive. Not many hernioplasties and appendectomies are performed at the Foundation Hospital; a good many are done at Charity Hospital. During a period of six months with approximately the same number of patients on the two services, there were nine pulmonary emboli at the Charity Hospital and only one at the Foundation Hospital. Naturally, this does not prove a great deal. It will require a long time to do that, but one thing is perfectly obvious: alpha-tocopherol is perfectly safe. It will not produce a bleeding tendency.

Because anticoagulants produce a bleeding tendency, we do not use them prophylactically. As a matter of fact, we use very few anticoagulants, because once the thrombosis has occurred, it does no good to use them. Anticoagulants will not prevent the detachment of the clot; therefore, the only thing to do is to apply a ligature between the clot and the heart.

If one could know which patient was going to develop phlebothrombosis, one might use anticoagulants preoperatively or prophylactically. I feel, however, that anticoagulants are far too hazardous generally, because of the danger of bleeding, to permit their routine use. For that reason we do not employ them. But alpha-tocopherol is safe.

OBSERVATIONS, FACTS AND RECOMMENDATIONS DERIVED FROM A MENTAL HEALTH PROJECT FOR THE DEAF

JOHN D. RAINER, M.D.
FRANZ J. KALLMANN, M.D.
NEW YORK, NEW YORK

BY INVITATION

WE appreciate this opportunity to report our observations in a research project concerned with the adjustment problems of the deaf to a group of specialists in the clinical aspects of hearing. Along with a brief review of the organization and preliminary findings of our pilot study in New York State, we should like to present to you a number of practical recommendations formulated mainly from a psychiatric point of view.

Our hope is that you, as an interested and, needless to say, highly competent professional committee, may be moved to endorse what amounts to a blueprint for a constructive mental health program for the deaf. We also hope that you will advise us of any changes in our program that you may deem desirable. And we hope that, as a consequence, you may wish to explore the possibility of developing similar programs in other parts of the country as well as in other groups of seriously handicapped persons deprived of modern psychiatric treatment methods.

From the Department of Medical Genetics, New York State Psychiatric Institute, Columbia University, 722 West 168th Street, New York 32, New York.

Presented in a *Special Program* of the Otosclerosis Study Group and Committee on Conservation of Hearing, at the Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12, 1958, Chicago. This is the tenth in a series of reports on the progress of the Mental Health Project for the Deaf, which is being conducted by the Department of Medical Genetics of the New York State Psychiatric Institute, Columbia University, aided by a grant from the Office of Vocational Rehabilitation of the U. S. Department of Health, Education, and Welfare.

Our Mental Health Project for the Deaf was activated three and a half years ago—on April 1, 1955, to be exact—under the sponsorship of the Office of Vocational Rehabilitation and with the most altruistic guidance of an interdisciplinary Advisory Council formed by prominent clinicians, educators and administrators familiar with the problems of the deaf. From the project's inception, Dr. Edmund P. Fowler, Jr., has been the representative of otology in this advisory group.[4]

The following general aims of the project were formulated:

1. To investigate the adjustive norms and particular family problems of the literate deaf in two statistically representative deaf populations, the total deaf population of the State of New York and all deaf twins in the eastern half of the United States.
2. To develop and apply improved methods for the psychiatric management of the specific problems of deaf adolescents and adults (over age 12).
3. To train specialized personnel for the management of the adjustive problems of the deaf.

An exploratory search for modern mental health services for deaf persons in the eastern states revealed few, if any, that were qualified to meet the urgent needs of this group. An equally critical lack was found in regard to

statistical and other scientific data on the emotional problems of deaf persons, the range of their adjustment levels under varying socioeconomic conditions, their general developmental needs, and the manner in which they dealt with family problems.[3,6,7]

It had long been realized that the adjustive and vocational potentialities of severely handicapped persons, such as the deaf, would have norms, dynamics and limits quite specific to their impairment. Clearly, the most effective way of dealing with such specific problems would be to prevent a crippling state of maladjustment by means of specialized guidance facilities, focused on prudent management of sex and family problems and fully equipped for early detection and active treatment of emotional disturbances that are easily disguised by overconformative trends in any minority group.[12,13]

However, as became evident in the earliest stages of our project, the number of deaf New York State residents in need of psychiatric help was much larger than had been anticipated on the basis of available information; no regional agency was sufficiently informed about the nature and magnitude of the services required; and specialized plans and facilities for these services were lacking everywhere, intramurally as well as extramurally.

In view of this unsatisfactory state of affairs, the immediate objective of our program was to combine the conduct of basic research with the development of centrally located guidance centers and the training of qualified personnel for these diversified tasks. Obviously, properly directed guidance depends on an understanding of all the problems existing in a certain segment of the population. In the case of the deaf, this requirement calls for a staff that can communicate with research subjects and clinic patients in a confidence-inspiring setting and also be sufficiently mobile to maintain prolonged contact with the families seeking guidance.[5]

The following subjects were among those to be investigated in our major psychiatric research project:

1. The usefulness of flexible methods of sex education in the presence of varying degrees of emotional immaturity
2. The effectiveness of such complex psychological mechanisms as sublimation, identification, and self-acceptance in the deprived state of a severe handicap
3. The need for a properly motivated system of occupational selection directed toward establishing steady working habits
4. The specific requirements for careful mate selection, family planning and marital compatibility in the face of lifelong conditions of frustration
5. The optimun standards for the upbringing of uniform or mixed sets of hearing or deaf children by one or two deaf parents.

For the study of these matters, a multidisciplinary research team of psychiatrists, psychologists, population statisticians and geneticists appeared indispensable. Fortunately, the task of assembling such a team was accomplished without undue delay.

At the same time, two days a week were set aside for the clinical part of the program—that of providing diagnostic, therapeutic and guidance services for literate deaf persons ranging in age from 12 to 42 years. These facilities were offered to deaf residents of the metropolitan district who were known

to have severe emotional difficulties or to be in need of advice with respect to important family affairs. All agencies and schools concerned with the deaf in this area were asked to refer suitable cases for diagnostic or therapeutic purposes. From the outset, the demand for these services was so active that, although several psychiatrists and psychologists were assigned to the management of clinic cases, a waiting list soon became necessary.[1]

Following the organization of clinic facilities at the Psychiatric Institute, elaborate plans were made for obtaining reliable research data on the total range of adjustive variations in the two deaf population samples chosen as representative of the entire deaf population of the United States (both rural and urban). The age group from 12 to 42 years was adopted for index cases (persons with early total deafness, whose personal and intra-family adjustment patterns were to be classified as poor, fair, or adequate according to levels of life performance).

For diagnostic ascertainment of single-born New York State residents, deafness was defined as a stress-producing hearing loss from birth or early childhood, rendering a person incapable of effecting meaningful auditory contact with the environment. Literacy, which was deemed an essential requirement for evaluating life performance levels of deaf index cases (the histories of illiterate research subjects to be recorded for statistical purposes only), was described as the ability to communicate meaningfully with the investigator, either verbally or by means of the sign language. The ten performance areas to be rated in literate research subjects were the following:

A. Regarding home life: performance as homemaker; spouse of a hearing or deaf subject; parent of hearing or deaf children, or both; child of hearing or deaf parents. B. Regarding community life: performance as worker capable or incapable of self-support; citizen; church member; club or association member; friend; participant in hobbies and other outside activities.

Generally speaking, the research aspect of the project was organized on the premise that systematic guidance can be provided for the deaf only if complete information is available on their adaptive capacities, as well as their specific types of adjustive difficulties. One of our guiding principles was that a true picture would have to be based on a survey of all deaf persons in a well-defined district, rather than on any kind of a biased sample.

Accordingly, during the earlier stages of the project the combined tasks of surveying the New York State population of deaf adolescents and adults and of collecting a representative sample of deaf twins in the eastern part of the country were carried out with the entire available personnel. By direct contact with national and state organizations, local agencies and clubs, church groups, schools for the deaf and all institutional systems, basic information was gathered on over 10,000 deaf residents of New York State, including many who had never been to school and were living on isolated farms or more or less anonymously in rural homes and institutions.

At present, the staff is about to complete the task of interviewing a random sample of the total population, using the whole range of communication techniques and applying a detailed scheme of exploring adjustment in all areas—health, education, vocation, family, and community. Life performance in these spheres is to be related to such factors as type of deafness and age at onset,

quality and extent of schooling, presence or absence of other deaf members in the family, preferred means of communication, criteria for choice of mate or reasons for intentional celibacy, and special problems of parenthood planning. It is hoped that this analysis will provide a sound basis for psychiatric guidance and therapy for the deaf.

To broaden the baseline of our general population study, our program includes a comparative investigation of twin pairs with one or two deaf members. Longitudinal data on their development, reflecting the differentiating effect of deafness, as well as genetic information of potential etiologic value, are expected to come out of this long-term study of twins and their families.[11]

An appraisal of data on the personal and family histories of grossly maladjusted members of the total deaf group —those at the lower end of the distribution of deafness-specific adjustment patterns—required another special survey which was organized with the aid of all New York State institutions for the mentally defective and disordered. The staffs were asked to prepare lists of deaf persons presently hospitalized or recently placed on convalescent status, and have subsequently been informing us of new admissions of deaf patients and those with deaf relatives. In this manner we obtained the clinical histories of over 500 deaf patients, leaving no doubt in our minds as to the need for a drastic reorganization of psychiatric services directed at rehabilitating a large proportion of hospitalized deaf persons. Rather than let them lie fallow and inaccessible to therapy in chronic wards, it seemed far more advisable to set aside specialized wards for deaf people in a few mental hospitals where they can profit from the care of specially trained personnel.

Towards this end various arrangements have been made by us to expedite the referral to our project of all deaf persons who reach a mental hospital, a penal or correctional institution, or an observation ward. Such a step seemed essential in view of the fact that we found many chronic cases with a long history of mental illness before admission. This situation alone indicated the need for some coordinated system facilitating early diagnosis and immediate treatment for psychiatric disorders of the deaf.[8]

For this express purpose, a senior member of our staff is assigned the task of seeing every newly admitted deaf patient in the metropolitan area as soon as a telephone report comes in. Following consultation with the hospital authorities, recommendation is made either for the patient's admittance to a certain institution or for his release and subsequent referral to our ambulatory treatment services.

In line with the observations made in our district, we have recommended that special services for mental health work with the deaf be established in at least every major population area. These facilities might be located at one of the larger State hospitals, where those deaf patients requiring hospitalization would receive the particular care they need. Instead of being dispersed in many hospitals, these patients would greatly profit from being together in special units set aside for them. Following their discharge, they would be assigned to a separate division of a convalescent clinic for aftercare. Such clinics could also provide the families of deaf patients with whatever guidance and supportive measures are needed. In addition, with a nucleus of doctors and other workers with knowledge of the deaf, the clinics would be available for ambulatory treatment

of emotionally disturbed deaf people requiring no hospitalization.

Several provocative sidelights have emerged from our observations on approximately 250 deaf mental hospital patients and a like number of persons in state schools for mental defectives. While our general population survey to date has yielded a preliminary rate of about 0.08 per cent deaf in the state, the rate of deaf persons in mental hospitals is three times as high, and in state schools for mental defectives, twenty times as high.[9]

This fact may have any one or all of three possible explanations:

1. Our hospital census may be considerably more complete at this point than our census of deaf persons in the general population.

2. The need for institutionalization may be greater among the deaf than among the hearing.

3. The average length of hospitalization of the deaf may exceed that of hearing patients.

The group of hospitalized deaf patients is of interest for various reasons. In a large number (25.6 per cent), the case was diagnosed as psychosis with mental deficiency. Had they been hearing persons, many of these patients might have been placed in a more precise psychiatric classification. As you know, the largest category, among the deaf as well as the hearing, is the schizophrenic group (47.5 per cent). For obvious reasons, however, the usual psychiatric criteria for diagnosing schizophrenic processes in terms of disordered thought and emotion are grossly obscured by communication barriers.[2]

In advanced stages, catatonic, hebephrenic and paranoid symptoms among the deaf show no significant difference from those observed in the hearing. Among the deaf with indeterminate behavior deviations, however, the diagnosis of schizophrenia poses a particular problem. Intensive genetic and developmental studies will be necessary to answer such questions as the following:

1. What are the signs of early or latent schizophrenia in the deaf, and do they fall into a definable pattern?

2. What is the significance in the deaf not only of behavior forms resembling mental defect but of impulsive or psychopathic syndromes?

3. Is there any reason for believing that deafness might protect a person against schizophrenia altogether, rather than induce it?

Up to now, our study has yielded no evidence for a significantly increased prevalence of schizophrenia in the deaf population of New York State. However, much additional work is needed to distinguish the types and overt clinical manifestations of mental illness among the deaf, and to identify the optimum degrees of adjustment found on different levels of intellectual capacity and personality integration.

The rest of this report will be devoted to a matter that is probably of particular interest to you; namely, the task of securing cooperation in a project such as ours through reality-grounded public information and education. Conducting research and providing health services for a special group are badly hampered unless the members of the group are themselves aware of the need for such work. Specifically as to mental health planning, it was apt until very recently to be blocked by the social stigma attached to any emotional illness. This tendency was found by us to play a particularly important part among the deaf. Had we allowed ourselves to become dis-

couraged because of it, very little would have been accomplished by our project.

Various initial obstacles, for instance, were encountered in constructing our frame; that is, in ascertaining the names of all deaf residents of the State of New York. As previously mentioned, no adequate information existed about their numbers, addresses or identities. It was quite a disappointment to discover that a state law requiring medical reporting of early total deafness had not been complied with for many years, not had any attempt been made to enforce it in some way. In fact, the law was about to be quietly repealed. Moreover, some of the schools for the deaf were hesitant about listing their alumni, one large religious organization withholding any active cooperation to this date. In each case, the stated feeling was that the deaf would misinterpret and resent any inquiry whatsoever into their private lives and mental health.

Nevertheless, the support of many organizations, schools and hospitals made it possible for us, within two years, to file the names of nearly 11,000 deaf individuals, arranged according to location and family. When the task of sampling and interviewing began, we observed that our workers were received in cooperative spirit by most of the persons called upon. Yet too often we found that our letter of explanation preceding the visit had been ignored, and in two large cities, our invasion in the local community was flatly considered suspect by the regional medical society and the Chamber of Commerce.

The need for public enlightenment was dramatically underscored when a questionnaire was sent to a group of 8,000 deaf adults in our files. We had thought by this means to verify current addresses, marital status, and family affiliations, to appraise the attitude toward special mental health services for the deaf, and to complete our sample of twins, which at that time showed a certain deficit. Close to 30 per cent of those who presumably received the questionnaire replied; of them, well over 80 per cent answered in the affirmative with respect to the need for better mental health services. However, the numerous unreturned questionnaires and other negative expressions that came to us by mail indicated that we had to embark upon an immediate educational campaign.

There was another reason for prompt action in this direction. Our clinical program (both intramural and extramural) had reached the point where it was apparent that the transition from experimental to regular services could not be made without the openly expressed demand for such services on the part of potential beneficiaries and their professional advisors.

Accordingly, the members of our Advisory Council drew up the following resolution last year:

> We are agreed that there is an urgent need for more adequate and more integrated mental health services for the deaf in the districts covered by this pilot project. The need extends to psychiatric services for both sexes (children and adults) on an inpatient and outpatient level (metropolitan and upstate districts) as well as to a properly coordinated vital statistics system for the deaf (preferably coordinated by an interdepartmental agency with the authority to unify the principles of reporting and recording).
>
> Prerequisites for this program are: (a) the availability of specialized professional personnel; (b) progressive attitudes toward the special adjustment problems of this subpopulation; and (c) the realization on the part of all agencies and organizations, dealing with these particular problems, that the deaf are entitled to the same quality and amount of mental health services as are available for the hearing.
>
> It is unrealistic and contrary to the best interests of the deaf to perpetuate the tendency

to minimize the need for an active mental health program for this group.

To further the understanding of these objectives by the public at large, a working conference was held for representatives of New York State organizations for the deaf. This meeting was well attended and proved to be successful beyond all expectations, as was shown in a documented brochure.[10]

At the close of the impressive conference, the delegates proposed the following four-point platform for further action:

1. The deaf need and are entitled to mental health services equal to those available for the hearing.

2. Specialized psychiatric and psychological services should be made available throughout the State.

3. To make these available on a permanent basis, the need must be made known through proper administrative and legislative channels, both State and Federal.

4. Organized steps should be taken by each group individually, as well as by all groups collectively, to promote this important objective.

These suggestions were officially adopted at the recent convention of the Empire Association of the Deaf, the largest organization of its kind in New York State. Towards this end, a mental health committee was formed, charged with enlisting the aid of administrative and legislative authorities. What was most gratifying was the fact that key persons who had been skeptical or even somewhat apprehensive of the program became enthusiastic supporters, to the delight and encouragement of those of their group who had been active from the start. Educators and officials working with the deaf for many years called this turn of events a unique milestone in the history of the deaf.

This is the point reached by the project. Naturally, a great deal of work remains to be done, not only in planning for the future, but also in sifting our research data. In the meantime, the clinical program continues to operate on a makeshift basis. The main thing is, however, that the rationale for a mental health program for the deaf has been demonstrated to the satisfaction of the group that is most concerned. As a blueprint for a constructive program, therefore, this has grown into an issue on which all interested parties will be able to unite.

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HISTOPATHOLOGIC CHANGES FOLLOWING FENESTRATION AND STAPES MOBILIZATION

JOHN R. LINDSAY, M.D.
CHICAGO, ILLINOIS

ANDERSON C. HILDING, M.D.
DULUTH, MINNESOTA

JAMES W. MCLAURIN, M.D.
BATON ROUGE, LOUISIANA

NELSON S. KEELER, M.D.
OAKLAND, CALIFORNIA

HOWARD M. HOUSE, M.D.
LOS ANGELES, CALIFORNIA

THE material for this report comprises five cases of bilateral otosclerosis. In four cases the patient has had a fenestration performed on one ear. In one case mobilization of the stapes has been carried out on both ears. One of the fenestration cases, contributed by Dr. Howard House, was the subject of a report given in 1950 before the Otosclerosis Study Group (House, Perret and Lindsay). In the three new cases treated by fenestration the temporal bones were eventually recovered by Dr. James McLaurin, Dr. Nelson Keeler, and Dr. Anderson Hilding. The case in which there was a bilateral mobilization of the stapes was contributed by Dr. House.

Histopathologic findings to be reported in each case will be limited mainly to the character and distribution of the otosclerosis, the extent to which the stapes has been involved and the findings related to the operative procedure.

Presented in a *Special Program* of the Otosclerosis Study Group and Committee on Conservation of Hearing, at the Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12, 1958, Chicago.

CASE 1

(Reported in Supplementum to Transactions of the American Academy of Ophthalmology and Otolaryngology, October 1950, pages 13-21, by House, Perret and Lindsay.) Female, age 47. Fenestration of the right ear for otosclerosis resulted in hearing gain averaging 15 db for the three speech frequencies (fig. 1). Accidental death by electrocution occurred two years after the operation.

Summary of Histopathologic Findings

A review of the histopathologic findings in this case revealed that a large focus of otosclerosis on each side extended from the oval window region to the round window niche. The review also disclosed that in the left ear, a small area of the footplate just below the anterior crus was invaded by the otosclerotic focus and a small area of ankylosis was present. The review did not reveal ankylosis on the right side, but it is probable that a limited patch of otosclerosis with ankylosis affected this side also. The previous report that ankylosis was not demonstrated and that the sta-

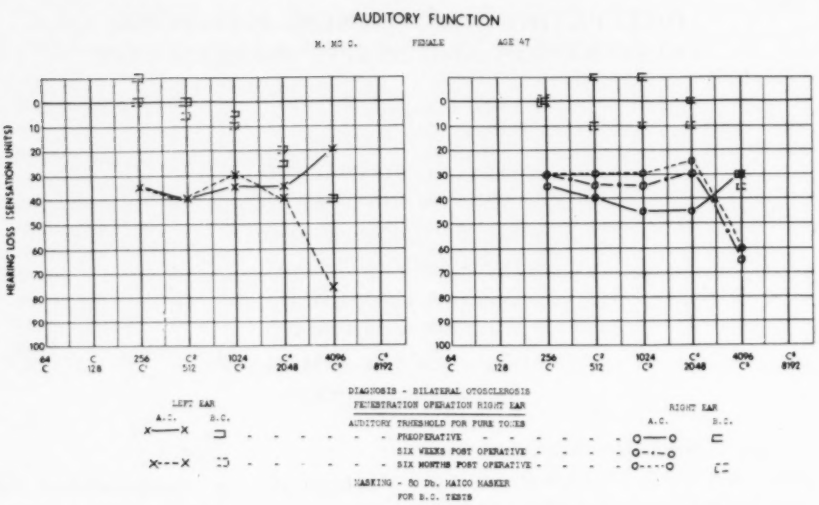

FIG. 1—Case 1. Female, age 47. Preoperative and postoperative pure tone thresholds.

FIG. 2—Case 1. Female, age 47. Section through the fenestra.

pes footplate was not involved by otosclerosis on either side proved to be inaccurate and was explained by technical difficulties which led to defects in the preparations. The labyrinthine capsule bordering on the annular ligament anteriorly and superiorly was replaced by otosclerotic bone in the active state. The ankylosis on the left was present only in a very small area.

Comment: The presence of otosclerotic changes in the footplates and the ankylosis in only a very limited extent indicated that both ears would have been suitable for mobilization. Mobilization in this type of otosclerosis would not present a difficult problem.

The conditions at the fenestra were good, including location, width, freedom

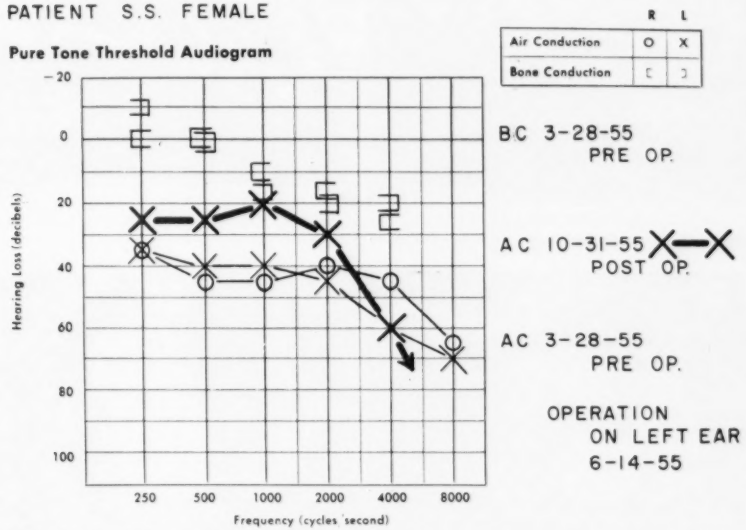

FIG. 3—Case 2. Preoperative AC and BC audiograms and postoperative AC audiogram (heavy line) of left ear, four and one-half months postoperative.

The round window niches were patent. The fenestra on the left was covered by a moderately thin skin flap (fig. 2). A thin layer of fibrous tissue had developed on the undersurface of the flap in the fenestral gap. No bone chips were present. The slightly flattened membranous canal lay against the outer part of the flap adjacent to the posterior bony margin.

from bone chips, and normal position of the membranous canal.

A small seam of new bone of apparent periosteal origin lay beneath the flap at the anterior margin but did not narrow the fenestra. The location of this new bone suggests that contraction of fibrous tissue along the undersurface of the flap may have caused a tendency

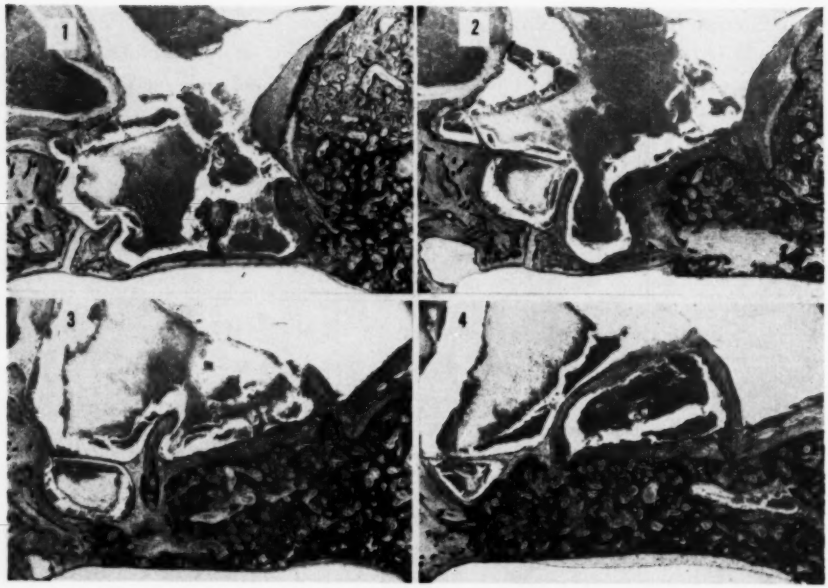

FIG. 4—Case 2. Left ear, four sections through the stapes. Anterior crus incorporated in a mass of otosclerotic bone extending onto the footplate. Posterior crus is ankylosed a little above its base.

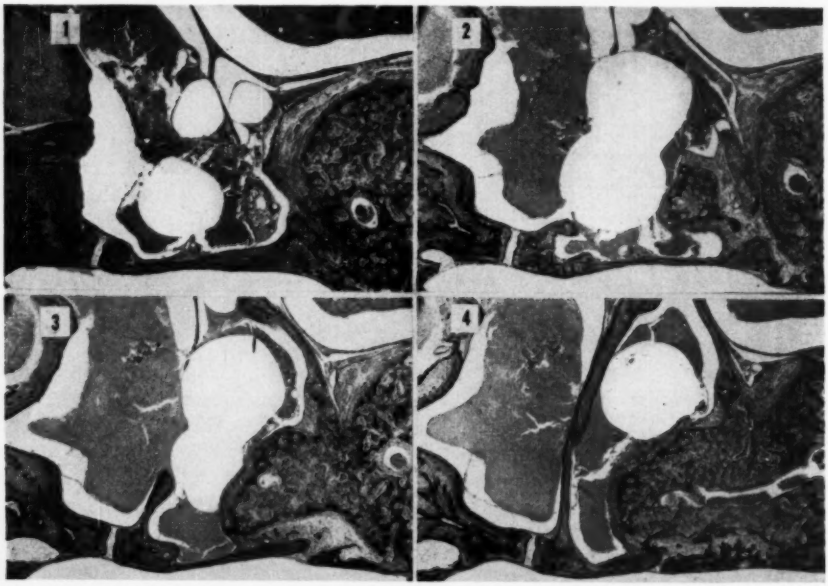

FIG. 5—Case 2. Right stapes, four sections showing a mass of otosclerotic bone extending onto the footplate fixing the anterior crus. The posterior crus and about two thirds of the footplate are free from otosclerosis.

of the flap to pull away from the bone, allowing new bone to form between. A fenestra placed on the convexity of the canal may predispose to better apposition at the edges.

CASE 2

Adult female. Accidental death five months and eighteen days after fenestration of the left ear. The last audiogram was made four and one-half months after operation (fig. 3).

The otosclerosis was limited to one large active focus in each ear extending from the cochlea and oval window region down to and almost surrounding the round window but not blocking it.

The footplate was involved by the otosclerosis on both sides to an extent estimated at about one third of its area.

On the left side (fig. 4), the anterior crus was incorporated in the mass of otosclerotic bone which extended onto the footplate. The base of the posterior crus, along with part of the footplate, was free of otosclerosis, but a little above its base the posterior crus also was invaded by the otosclerotic process and had ankylosed to the promontory.

On the right side (fig. 5), the thick vascular mass of otosclerotic bone extended from the anterior margin onto the footplate. The anterior crus was otosclerotic and incorporated in the diseased bone. The posterior crus was normal along with about two thirds of the footplate.

Certain comments seem justified regarding the histopathologic indications as to the possibility for successful mobilization of the stapes in ears such as these.

A. *Left stapes.* Certain features of the left stapes would make a successful mobilization difficult to achieve.

The thickness and vascularity of the otosclerotic focus would make mobilization by chiseling or breaking through the focus inadvisable. Immediate success by such a procedure would probably be followed by re-ankylosis.

Mobilization by fracture through the unaffected part of the footplate and any attempt to free the posterior crus from the otosclerotic mass would also be likely to fail because of the probability of a fracture of the crus, or if separation from the main mass were achieved, re-ankylosis would be likely.

B. *Right stapes.* It is apparent that mobilization by cutting or breaking through this thick otosclerotic mass would be difficult to achieve and probably would be followed by re-ankylosis.

Fracture through the footplate and anterior crurotomy would be suitable if it can be assumed that a fracture of the footplate will heal only by fibrous union.

Fenestration Site

Some features of the fenestral site may be indicated as of particular significance (fig. 6). The covering flap of skin was quite thick and edematous, yet the hearing gain was fairly satisfactory. The edema was explained by the fact that this cavity had not yet healed completely after five months and eighteen days.

There was a layer of new fibrous tissue on the undersurface of the flap. In part of the fenestral gap the membranous canal lay in its normal position in contact with the flap. As the posterior end of the fenestra was approached, the membranous canal was displaced by a few bone chips lying beneath the bony margin. The fibrous layer between flap and canal was thickened and continuous, with fibrous tissue extending from the endosteum on either side. Much new

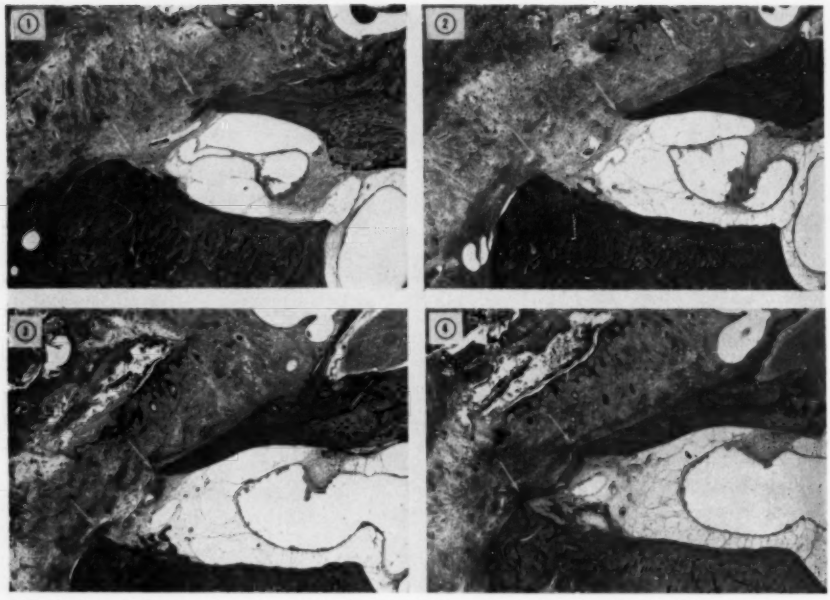

FIG. 6—Case 2. Four sections through the fenestra (white arrows). Covering flap is thick, edematous and not completely epithelized. Hemorrhage in the flap and in the labyrinth due to head trauma. Considerable fibrosis and some osteogenesis in the perilymphatic space. Five and one-half months after operation.

bone had been deposited in this fibrous tissue, extending from the endosteal bony surfaces. Only a few spicules of old bone were seen. Most of the fibrosis and bone formation originated on the endosteal bone surface, probably as a result of trauma during removal of chips.

Osteogenesis in the perilymphatic space was still in an active stage; however, the major part of the fenestral gap showed a normal relationship of membranous canal and fenestral covering without signs of osteogenetic activity. The fenestra would in all probability have remained open at the anterior end. Regeneration of bone had not reached the endpoint in the perilymphatic space at and just beyond the posterior end. The membranous canal was narrowed in the area where this fibrosis was prominent but was not obliterated. A corresponding finding has been observed in experimental fenestrations in monkeys.

CASE 3

Female, age 60. Bilateral otosclerosis. Right fenestration in 1953. Last audiogram available in November 1955. Hearing improvement remained satisfactory (fig. 7). Death on January 20, 1958, resulted from a motorcycle accident four days earlier.

The histopathologic findings will be reported in detail at a later date. They include significant changes which are related to the head trauma. The present comments are limited to the type and

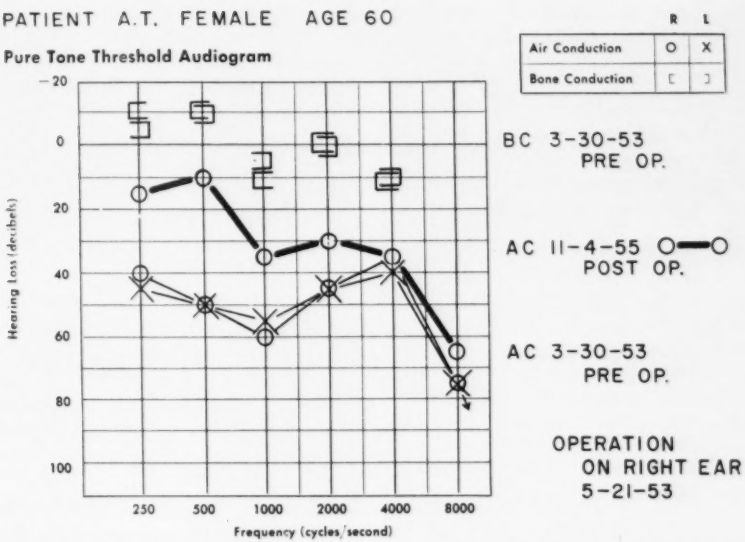

FIG. 7—Case 3. Preoperative audiograms AC and BC. AC audiogram (heavy line) made two and a half years after operation.

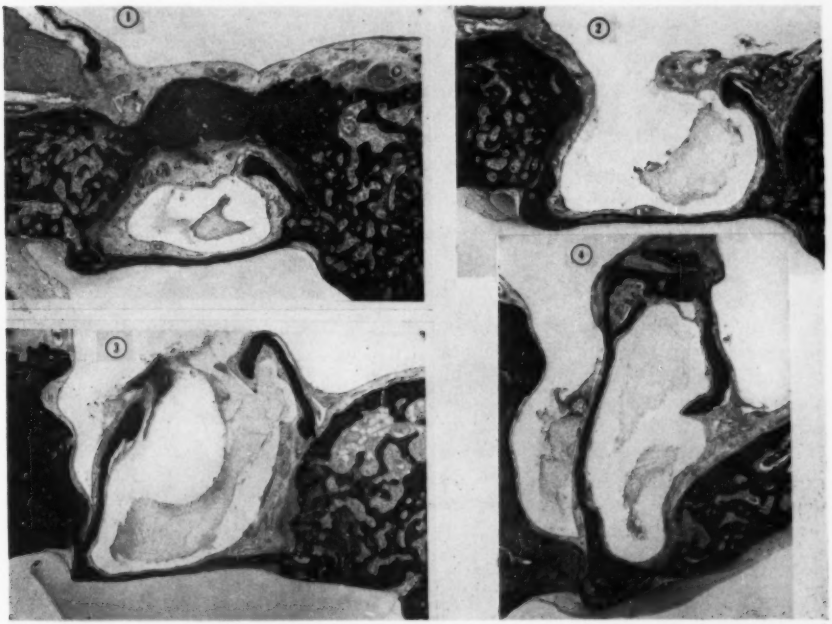

FIG. 8—Case 3. Four sections through left stapes. Ankylosis has occurred along the superior and anterior margins, and the anterior part of the lower margin. Posterior edge of the stapes footplate is subluxated.

extent of the otosclerosis, the condition of the stapes on each side and the fenestration on the right.

There was one large active focus of otosclerosis on each side between the oval window and the cochlea, extending down the promontory to the attachment of the round window membrane.

In addition, there were smaller patches of otosclerotic bone bordering chiefly on the semicircular canals on either side.

The left stapes (fig. 8) was ankylosed across the superior and anterior margins and in the anterior part of the lower margin. The anterior crus was composed of otosclerotic bone at its junction with the footplate. The posterior crus was free from otosclerosis except at the upper margin of its base, where ankylosis had begun. The footplate was free from otosclerosis except along the upper and anterior margins and the anterior part of the lower margin, and at these margins there was ankylosis.

Comment: Mobilization of the complete stapes in this case probably would have been difficult because of the extent of the ankylosis and the involvement of the anterior crus. Re-ankylosis would be probable.

A fracture through the footplate and an anterior crurotomy would appear preferable in this case although since only a small part of the circumference was free of ankylosis, a comminuted fracture of the footplate could be expected. The duration of mobility would depend upon whether such fractures healed by fibrous or bony union.

Right stapes (fig. 9). Ankylosis extended around the full circumference except for small areas anteriorly and posteriorly.

Comment: Mobilization, whether of the whole footplate or only a part (bipodal or monopodal), would involve fracturing through otosclerotic bone in a state of activity. A predisposition to re-ankylosis could be expected.

Fenestration Site

The fenestral gap was patent almost to the original width throughout most of its length (fig. 10).

The membranous canal was in its normal position in contact with the flap except at the ends of the fenestra. The skin flap was of moderate thickness. On its undersurface a layer of fibrous tissue could be identified extending across the gap between membranous canal and skin flap. At each side of the membranous canal this fibrous lining of the flap was continuous with endosteum and periosteum at the point of meeting. There was no new bone in this fibrous layer except at certain limited areas. One area was around a small chip of bone which had depressed the membranous canal at the anterior margin of the fenestra where the bone edge was thick; another, at the ends of the fenestra where the membranous canal was depressed by bone fragments which had slipped beneath the bony margin and where endosteum had been torn loose for a short distance.

There was extensive recent hemorrhage in the perilymphatic space. The flap was edematous and contained much extravasated blood.

Comment: The end result of the fenestration procedure was a permanently healed fenestral gap which had maintained almost its entire size. The membranous canal lay in its normal position against a covering flap which was free from new bone except at the extreme ends, where small bone chips had been left, causing distortion, and at one side where the bony margin was thick.

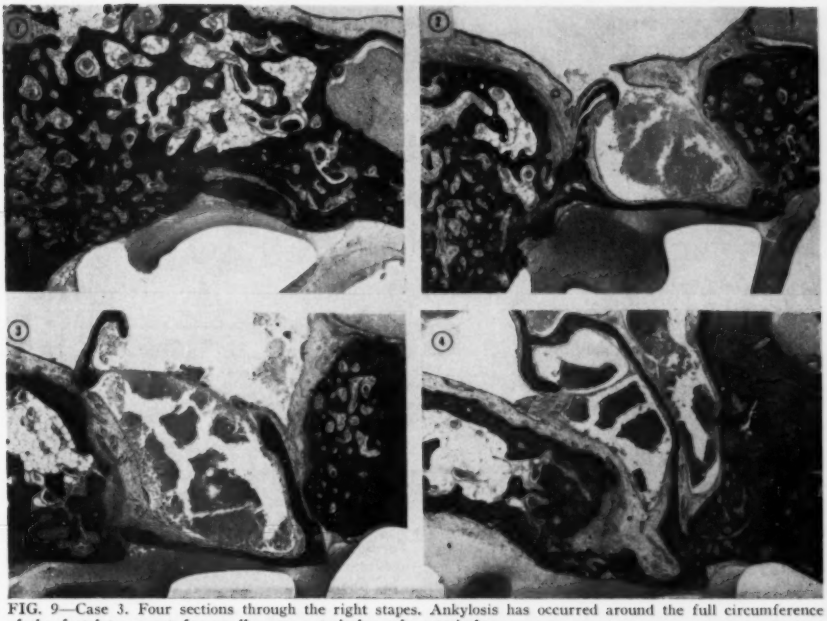

FIG. 9—Case 3. Four sections through the right stapes. Ankylosis has occurred around the full circumference of the footplate except for small areas anteriorly and posteriorly.

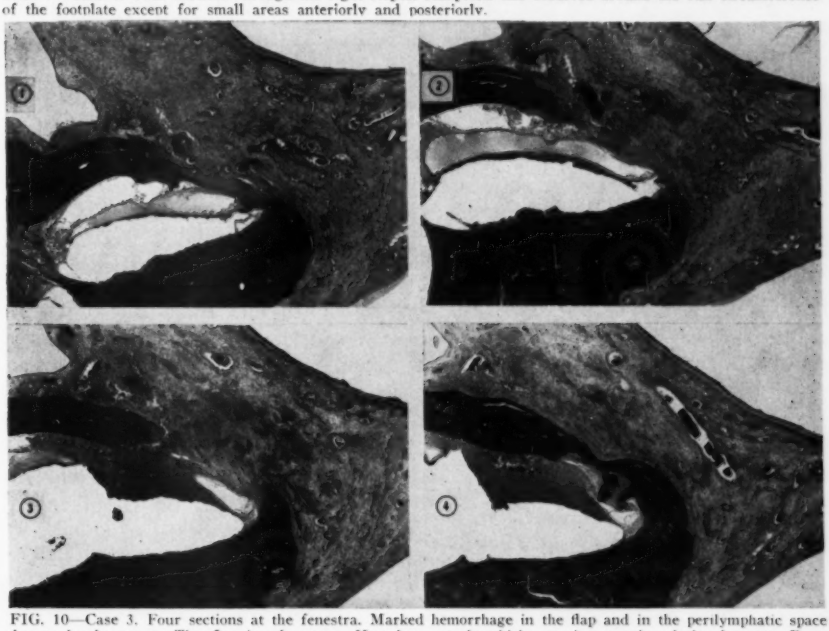

FIG. 10—Case 3. Four sections at the fenestra. Marked hemorrhage in the flap and in the perilymphatic space due to head trauma. The flap is edematous. New bone at the thick anterior margin of the fenestra. Bone fragments beneath the posterior margin. Fenestra is widely patent.

CASE 4

Female, age 59. Bilateral otosclerosis. Cause of death: uterine malignant lesion.

A right fenestration had been done in 1949. Audiograms made preoperatively and six and one-half years postoperatively are shown in figure 11.

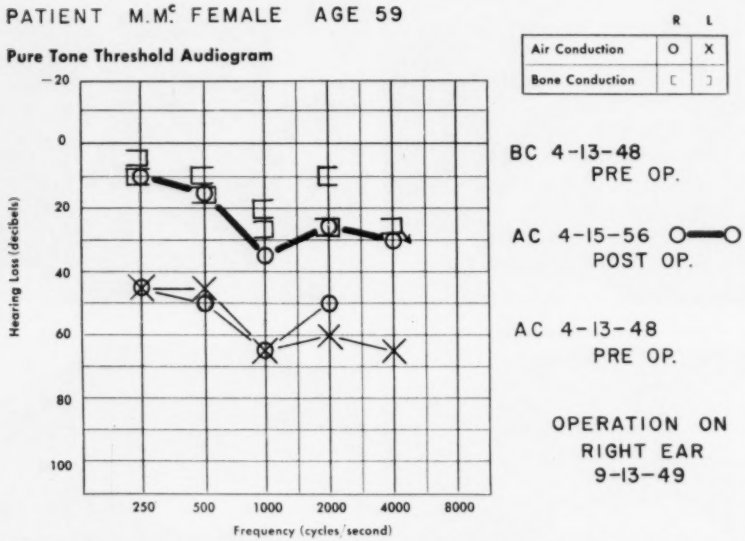

FIG. 11—Case 4. Preoperative AC and BC audiograms. Postoperative AC audiogram on the right ear (heavy line) made eight years after operation.

Death occurred two weeks after the last audiogram was made. This report is limited to the type and extent of the otosclerotic lesions, the condition of the stapes and labyrinthine windows and the surgical fenestra.

Left ear. One focus of otosclerosis was present, partly sclerotic but partly vascular and apparently in an active state. It was located at the usual site between the cochlea and the oval window. The focus did not extend to the round window margin.

The footplate of the stapes was composed of otosclerotic bone throughout (fig. 12). It was several times the normal thickness. The posterior crus was composed of otosclerotic bone; it was greatly thickened almost to the point of junction of the crura. The anterior crus was thin but composed of otosclerotic bone in its lower half. The bone in the footplate and around the oval window contained many vascular spaces showing signs of active bone resorption and replacement. These were interspersed with areas where the bone was sclerotic and contained narrow spaces in which there was a vessel but relatively few cells around it. The footplate was ankylosed to the labyrinthine wall throughout its entire circumference.

Right ear. One focus of otosclerosis was found at the oval window region.

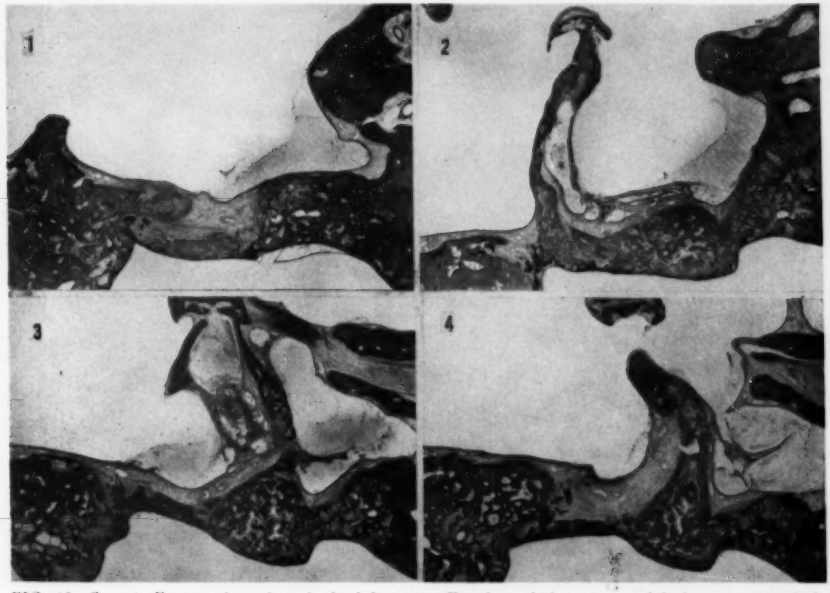

FIG. 12—Case 4. Four sections through the left stapes. Footplate of the stapes and both crura extensively involved by otosclerosis. The footplate was ankylosed throughout the entire circumference.

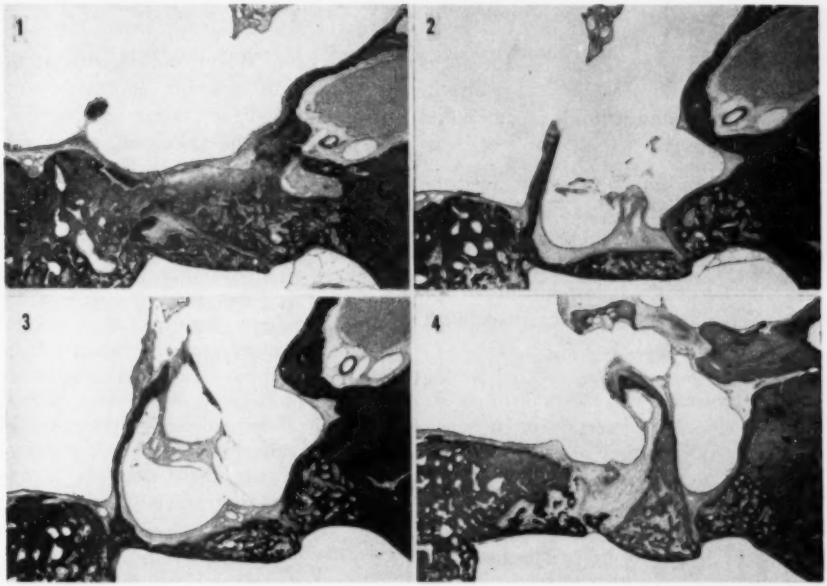

FIG. 13—Case 4. Four sections through the right stapes. The footplate and the posterior crus show extensive involvement by otosclerosis. Ankylosis has occurred throughout almost the entire circumference.

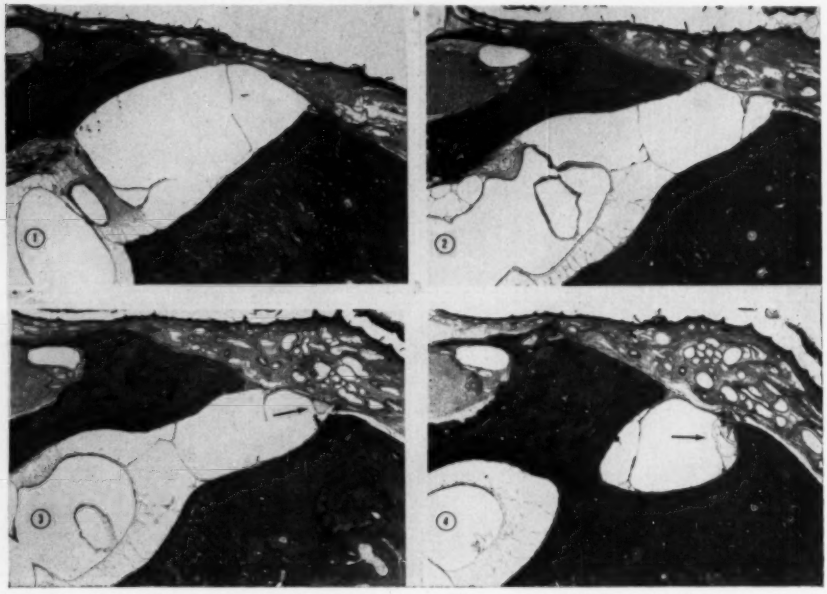

FIG. 14—Case 4. Four sections through the fenestral gap. The gap is completely patent. The flap is thin. A few bone chips are seen at the posterior margin. The membranous canal is missing throughout the length of the fenestra. The beginning of the sealed off membranous canal at the distal margin of the fenestra is indicated by arrows. Satisfactory hearing improvement.

There was a prolongation of the focus in the promontory as far as the attachment of the round window membrane at the outer margin.

The otosclerotic focus surrounded the oval window entirely and had replaced all of the footplate except a very small portion bordering on the margin near the anterior crus, where remnants of the original bone and calcified cartilage were still present (fig. 13).

The footplate was several times its normal thickness except for a small area near the anterior crus. The posterior crus was many times its normal thickness at its base, tapering down towards the neck. The anterior crus was normal except at its base. The footplate was ankylosed almost throughout its circumference.

Comment: When considered from the viewpoint of possible successful mobilization, two factors are unfavorable: the extent of the ankylosis, which was almost complete on both sides, and the composition of the bone through which any mobilization procedure would have to be carried out. Evidence provided by the case of mobilization to be reported later in this series, as well as increasing clinical evidence, indicates that ankylosis has a tendency to recur when a fracture or chisel cut is made through thick vascular otosclerotic bone. Any method of mobilization of these footplates would involve breaking through thick otosclerotic bone; hence, there would be a predisposition to recurrence of ankylosis.

Fenestration Site

The fenestral gap on the right side was patent to the maximum width and

was covered by a thin skin flap which on its inner surface consisted of a thin fibrous layer continuous with the endosteum and periosteum at the margins (fig. 14).

At the distal end of the fenestral gap a few small spicules of bone were depressed beneath the bone, distorting the membranous canal at that point. New bone had been laid down to a limited extent in that area.

A finding of particular interest in this case was that the membranous canal was missing throughout the fenestral gap region. The canal was interrupted near the ampulla and the ampullary end involuted back into the ampulla, while the distal portion was found to begin near the posterior end of the fenestra. The canal was obviously sealed off at the end of this distal segment and whether it was collapsed at the torn ampullary end was not certain.

Comment: Certain important features are illustrated by the fenestra in this case. The fenestral gap was clean and free from bone chips and the bony margins of the gap were thin, factors which have been observed to favor the prevention of osteogenetic closure. The flap was quite thin in the region covering the fenestra. The absence of edema in this flap, as compared to cases 2 and 3, explains in part the great contrast in thickness.

Of special interest was the fact that the fenestral gap had remained completely patent and the hearing had remained at a satisfactory level despite the injury and the break in the continuity of the membranous canal.

CASE 5

Female, age 45. Bilateral otosclerosis. Preoperative and postoperative air and bone conduction thresholds are shown in figure 15. Death occurred February 10, 1958, apparently from suffocation.

A stapes mobilization had been done on the left ear on January 21, 1957, twelve months and nineteen days before death, also on the right ear on June 18, 1957, seven months and twenty-three days before death. The operations were performed by Dr. House.

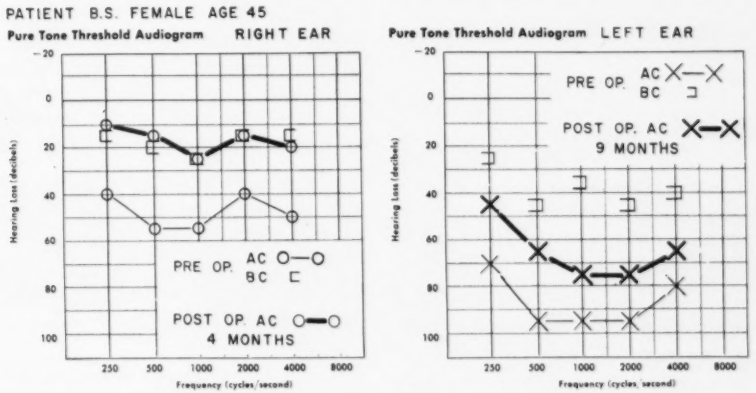
PATIENT B.S. FEMALE AGE 45
Pure Tone Threshold Audiogram RIGHT EAR
Pure Tone Threshold Audiogram LEFT EAR
Hearing Loss (decibels)
Frequency (cycles/second)
PRE OP. AC BC
POST OP. AC 4 MONTHS
POST OP. AC 9 MONTHS
250 500 1000 2000 4000 8000

FIG. 15—Case 5. Preoperative and postoperative audiograms. Stapes mobilization bilateral.

The operative notes are as follows:

Left ear: Unable to mobilize through the head. Grade I difficulty in mobilization at the footplate with needles. Grade II mobility obtained. No loss of perilymph or tear of the ligament. No tinnitus; no crural fracture; no dislocation of the incus; no damage to stapes head.

Right ear: Grade II difficulty. Mobilized through the head of the stapes. No footplate work. Grade II mobility. No loss of perilymph; no nausea or vomiting; no ligament tear; no tinnitus; no crural fracture; no dislocation; no splitting of head of stapes.

There was no dizziness or postural dizziness at any time during or after the operation.

Histopathology

Left ear: The left temporal bone had been pneumatized only in the mastoid portion. There were no pneumatized cells in the perilabyrinthine or in the apical parts of the pyramid.

The middle ear showed a few thin fibrous adhesions. The tympanic membrane and the malleus were absent from the specimen. The incus was present and free of otosclerosis. The state of preservation of the sensory structures was poor. This was partly due to postmortem changes and partly due to fixation artifacts.

There was moderate decrease in the number of ganglion cells and nerve fibers in the basal coil of the cochlea, but apparently a normal number in the upper basal, middle and upper coils. The otosclerotic changes in the labyrinthine capsule were extensive.

Patches of otosclerotic bone were dispersed throughout the capsule. Sclerotic extensions were present, bordering on all three semicircular canals. A large focus, mostly sclerotic, bordered on the internal meatus. Another lay between the cochlea and internal carotid canal. The largest and most active area was anterior to the oval window, extending down to the round window.

The round window niche contained much dense fibrous tissue and the membrane was retracted toward the middle ear (fig. 16), but there was an irregular air column in the niche extending from the hypotympanum to a moderately thickened fibrous round window membrane.

The footplate of the stapes was completely replaced by otosclerotic bone (fig. 17) except for microscopic remnants of calcified cartilage at the posterior margin.

The bony capsule surrounding the oval window was composed of otosclerotic bone except for a small area on the posterior margin.

The posterior margin of the stapes was subluxated into the vestibule to the extent of its own thickness. Fibrous tissue repair of the annular ligament had occurred. Within this fibrous tissue, new bone had been deposited, indicating beginning bony ankylosis.

At the anterior margin, the otosclerotic bone of the capsule and of the footplate was irregular with dense fibrous tissue and osteoid tissue replacing the annular ligament. New bone had been formed in apposition to the older otosclerotic bone, and osteoid tissue was evident in many areas (fig. 18).

It was evident that the process was well advanced toward re-establishment of ankylosis.

The otosclerotic bone in the footplate was thick and showed evidence of active absorption and rebuilding of bone.

Comment: A feature in this ear which is worthy of special comment was the widespread distribution of otosclerotic

FIG. 16—Case 5. Section showing the round window niche which is partly filled with fibrous tissue. The round window membrane is retracted outward forming a pocket in which lies degenerating blood cells (arrow), probably a terminal manifestation.

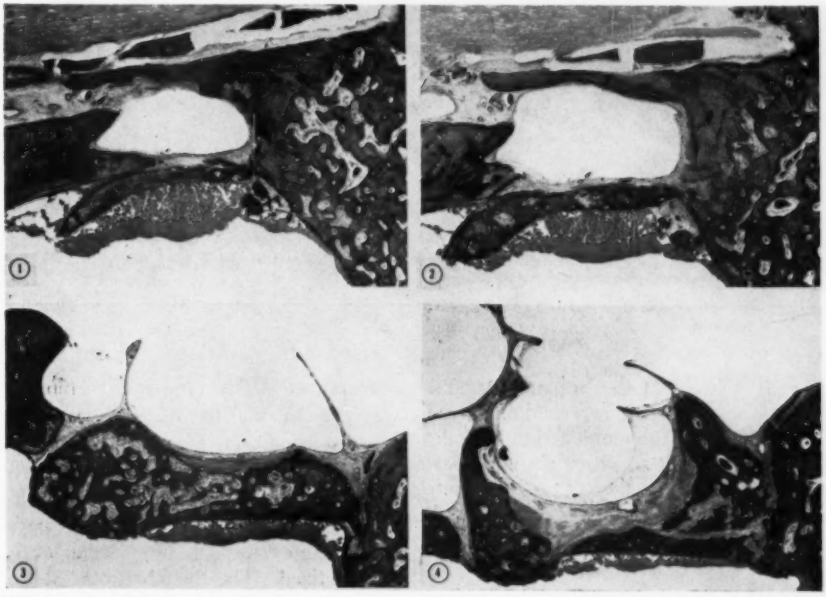

FIG. 17—Case 5. Four sections through the stapes. Degenerating red blood cells lie beneath the stapes, probably a terminal manifestation. The footplate has been subluxated posteriorly beyond the amount customary in cases of stapes ankylosis. The footplate and the bases of bone crura are composed of thick otosclerotic bone. Re-ankylosis beginning at the anterior margin of the footplate is shown in high power magnification in figure 18.

bone, mostly in the quiescent sclerotic stage, but in the stapes definitely active. There was evidence of bone regeneration in those areas in which fracture had gone through the otosclerotic bone at the time of mobilization. Also, new bone had been deposited at the point where the annular ligament had apparently been injured and healed by new fibrous tissue. These findings demonstrate a tendency for reformation of the ankylosis when the mobilization has taken place through an active focus of otosclerosis. The otosclerosis in this footplate would preclude any "mobilization" procedure except through thick otosclerotic bone.

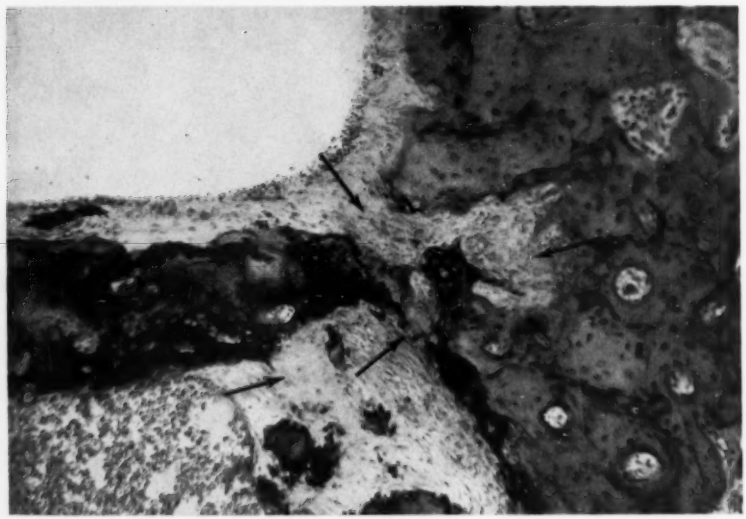

FIG. 18—Case 5. Anterior margin of the footplate showing osteoid and new bone formation (arrows) producing recurrence of ankylosis.

The fibrosis in the round window niche and the distortion of the round window membrane require explanation. The presence of otosclerosis in the bone bordering on the niche does not seem to account for these findings. That it had an inflammatory origin in early years is a more plausible explanation. The limited pneumatization of the left bone as compared to the right also is an indication of an inflammatory process in early childhood.

Right ear: The tympanic membrane was present, and the malleus and incus in good condition, free from otosclerosis. The apex of the pyramid and nearly all of the cochlea were missing from the specimen. The mastoid and the perilabyrinthine parts were extensively pneumatized. The membranous structures showed moderately advanced postmortem changes but were better preserved than in the opposite ear, in which

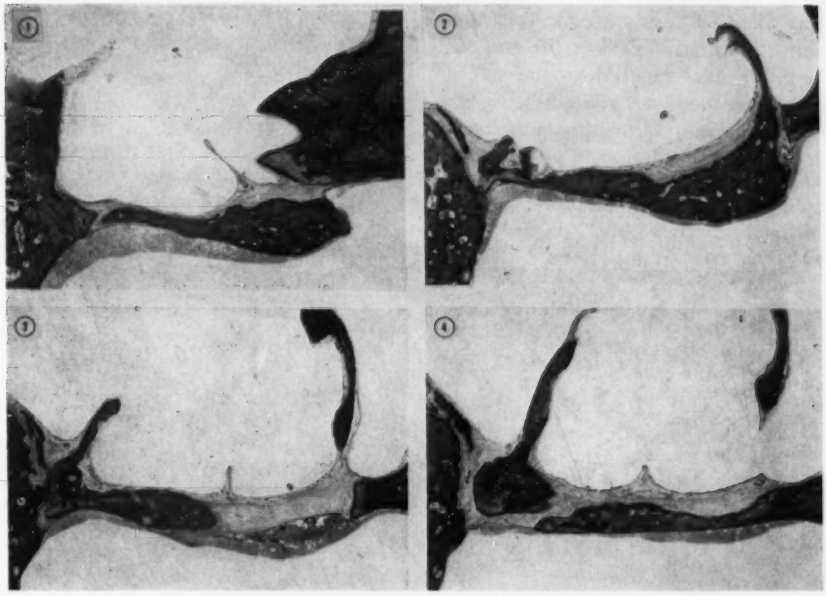

FIG. 19—Case 5. Four sections through the footplate of the right stapes. Subluxation has occurred at the posterior margin. At the anterior margin, lower left, new bone has formed in the annular ligament (arrow). A narrow dehiscence of the footplate is also seen extending from the posterior margin to the middle region (lower left). Footplate is extensively otosclerotic.

the inner ear spaces had not been opened during removal of the bones. Within the vestibule, bordering on the stapes footplate region, was a collection of red blood cells in an advanced stage of degeneration.

The otosclerotic foci in this bone also were dispersed irregularly throughout the labyrinthine end, confined mainly but not entirely to the enchondral layer of the capsule. There was a large focus at the internal meatus and a large focus in front of the oval window extending down in the promontory almost to the round window, but the round window was free from the disease.

The footplate of the stapes (fig. 19) was extensively involved by otosclerosis and was several times the normal thickness in the posterior half. The anterior margin was free from otosclerosis in the region close to the base of the anterior crus.

The upper border of the footplate was subluxated under the edge of the window margin. The anterior end was displaced outward and the posterior end inward. The posterior end of the stapes was most extensively otosclerotic, but the opposing posterior bony margin of the window was normal; whereas, at the anterior end of the footplate the bony margin of the capsule was otosclerotic and the margin of the footplate normal.

There was a gap in the bone of the footplate at its posterior end. The gap was narrow but extended for one half of the length of the footplate. This gap

was filled by fibrous tissue continuous with the annular ligament. At one point in the annular ligament at the anterior end of the footplate, where it had apparently been injured, new bone had been deposited and had already produced early ankylosis (fig. 20).

the circumference new bone had not formed.

The footplate of the stapes, although extensively involved, was normal along one margin. Also, the margin of the oval window was normal throughout half or more of its extent.

FIG. 20—Case 5. High power magnification of the area indicated by arrow in figure 19, lower left. This shows the new bone which is producing ankylosis between the anterior margin of the footplate and the otosclerotic margin of the labyrinthine capsule.

Comment: Features of significance in this ear in which the stapes had been mobilized with significant hearing improvement can be listed as follows:

The stapes was tilted out of its normal position and had healed in that position.

New bone had been laid down in the annular ligament at one point indicating a tendency for repair by osteogenesis with production of an early stage of ankylosis. Throughout the remainder of

Much of the otosclerotic part of the footplate lay opposite a normal oval window margin, and the otosclerotic part of the capsule lay opposite a normal part of the footplate.

These features probably accounted for a limited extent of ankylosis and favored successful mobilization.

The narrow bony dehiscence in the footplate could only be interpreted as a congenital defect.

SUMMARY

The five cases of otosclerosis with ankylosis of the stapes illustrate the great variation in the disease, the extent to which the stapes has been involved and the extent of the ankylosis.

They illustrate some of the variations in the problem which they would present if a mobilization procedure were undertaken.

The four examples of a fenestra bear out certain principles which have been indicated both on animal experimentation and from clinical experience on the human: The placing of the fenestra on a convex surface to promote closer adherence of the flap. Removal of all bone dust and fragments. Avoidance of stripping endosteum from the bone at the margins of the fenestra. The advantage of having a sharp edge to the fenestra to obtain close approximation between endosteal margin and the undersurface of the flap. One case illustrated that in the presence of a well prepared fenestra, a tear and gap in the membranous canal may be compatible with a good result.

Three of these four fenestration cases demonstrate the permanency of the well prepared fenestra and the improvement in hearing in suitable cases. The fourth case had not yet reached the end point of healing, but this case also showed a widely open fenestra after five and one-half months and a satisfactory hearing improvement. Maintenance of a patent fenestra could be expected in this fourth case although the perilymphatic space would be reduced at the distal end.

The case of bilateral stapes mobilization presented two most significant findings: The fracture through the active otosclerotic focus showed early re-formation of the ankylosis. Injury to the annular ligament, probably from subluxation of the footplate, was followed by osteogenesis at the site of injury with evidence of early ankylosis at one point.

ANTIBIOTICS AND THE INNER EAR

JOSEPH E. HAWKINS, JR., PH.D.
NEW YORK, NEW YORK
BY INVITATION

ALTHOUGH the antibiotic age, not yet two decades old, has largely freed the otologist from his former preoccupation with the surgical treatment of acute infections and has thus permitted him to turn his attention more and more to the study and relief of deafness, it has brought with it a new problem which is very properly the concern of the Committee on the Conservation of Hearing. This is the deplorable and yet fascinating property of *ototoxicity,* which is characteristic of a small but important group of antibiotics: streptomycin, dihydrostreptomycin, neomycin, viomycin and the new kanamycin, all of which are among the most effective agents against *Mycobacterium tuberculosis.* Historically and therapeutically the most important member of the group is streptomycin, the isolation of which was first announced by Waksman and his associates in 1944.[43] According to acute and chronic toxicity tests in laboratory animals, this substance, although obviously in rather an impure form, was well tolerated and relatively innocuous. It was therefore promptly submitted to clinical trial in the treatment of tuberculosis.

From the Department of Otolaryngology, New York University-Bellevue Medical Center, New York.

Presented in a *Special Program* of the Otosclerosis Study Group and Committee on Conservation of Hearing, at the Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12, 1958, Chicago. This is the tenth in a series of reports on the progress of the Mental Health Project for the Deaf, which is being conducted by the Department of Medical Genetics of the New York State Psychiatric Institute, Columbia University, aided by a grant from the Office of Vocational Rehabilitation of the U. S. Department of Health, Education, and Welfare.

STREPTOMYCIN

The first report of the use of streptomycin in human tuberculosis was published in 1945 by Hinshaw and Feldman.[25] They described it as a drug of low toxicity, producing relatively unimportant reactions of pain on injection, moderate elevation of body temperature, histamine-like responses of headache and flushing of the skin, mild malaise and a few instances of sensitization. Among the 34 patients treated by Hinshaw and Feldman, however, three developed an apparent disturbance of vestibular function, and one a transient deafness. These symptoms were attributed tentatively to a selective neurotoxic effect on the eighth cranial nerve. It was by no means clear whether the antibiotic itself or some impurity was at fault, since the preparations available probably contained less than 50 per cent pure streptomycin.

Further use of streptomycin by various investigators soon showed that the "neurotoxic" property discovered by Hinshaw and Feldman was characteristic of many, if not all, streptomycin preparations, provided they were administered parenterally for a period of weeks. Both the vestibular and the auditory disturbances might occur, although the former was predominant.

The Vestibular Disturbance

A number of authors have described and analyzed the vestibular disturbance in man caused by streptomycin, particularly Fowler and Seligman,[13] Glorig and

Fowler[15] and Northington.[36] The time of onset varied with the size of the dose. In Northington's series all patients receiving 2.5 to 3.2 gm. daily showed vestibular effects, usually during the second week of administration, while patients receiving 1.0 gm. daily were almost all symptom-free, even after seventy-five days of treatment. Glorig and Fowler's 32 patients received 1.8 to 2.0 gm. daily.

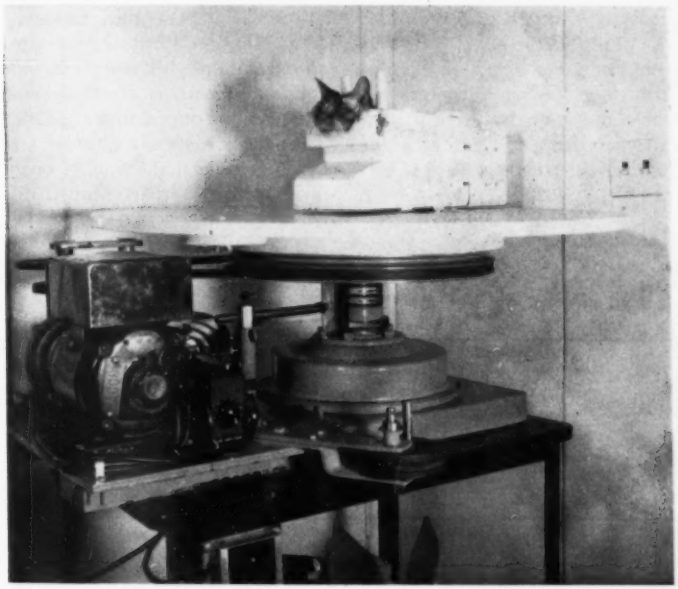

FIG. 1—Turntable for electrical recording of nystagmus in cats. The cat's head can be covered to eliminate the optokinetic component of the nystagmus. (Designed and constructed by M. Kniazuk in the Merck Institute for Therapeutic Research.)

Symptoms began on the eleventh day at the earliest, and within sixty days at the latest; six patients failed to show any symptoms in this period.

According to Glorig and Fowler, the vertigo produced by streptomycin is of an unusual type. Patients complained that when they turned their heads the sensation of turning persisted longer than the actual turning. Furthermore, one of the earliest symptoms noticed by the patients was blurring of vision while reading. Northington's patients also complained about a sense of false motion and a "jumbling" of distant visual objects. Spontaneous nystagmus was not seen, except in rare instances on lateral fixation.

The effect on posture and gait was similar to the acute effect of alcohol. Patients most severely affected were unable to stand or walk unaided. This was particularly true on a grade, an uneven surface, in the dark, or in an open field.

Three of Glorig and Fowler's patients showed a complete loss of response to caloric stimulation, even to 80 cc. of ice water. However, all of them had either vertigo alone or vertigo and nystagmus when subjected to galvanic stimulation, a finding which suggested that the strep-

tomycin lesion may be located in the peripheral vestibular end organs.

A disturbance of posture and gait in experimental animals treated with streptomycin was first described by Molitor and his associates,[34] who used dogs. They attributed the loss of equilibrium to a labyrinthine or cerebellar disturbance. Similar effects were observed in rabbits and cats treated with streptomycin.[16,17]

The cat is an especially favorable animal for the study of the action of streptomycin on vestibular function because of its highly developed righting reflexes, which enable the animal always to land on its feet when dropped from a height. With streptomycin treatment this ability may be completely lost.

Vestibular function in cats receiving streptomycin was tested at frequent intervals by recording the horizontal nystagmus in response to rotation on a motor-driven turntable (fig. 1). The corneoretinal potential was picked up by means of solder-disc electrodes pasted to the shaved skin on either side of the eyes near the outer canthi. Variation in the potential as the eyes moved from side to side was recorded, after suitable amplification, on an ink-writing oscillograph.[19,33,45] By this method it was found that an abrupt decline in the rate and duration of nystagmus induced by rotation begins at about the same time that minimal ataxia first appears (fig. 2). The larger the daily dose of streptomycin the earlier is the time of onset of this disturbance. With continued dosing, all

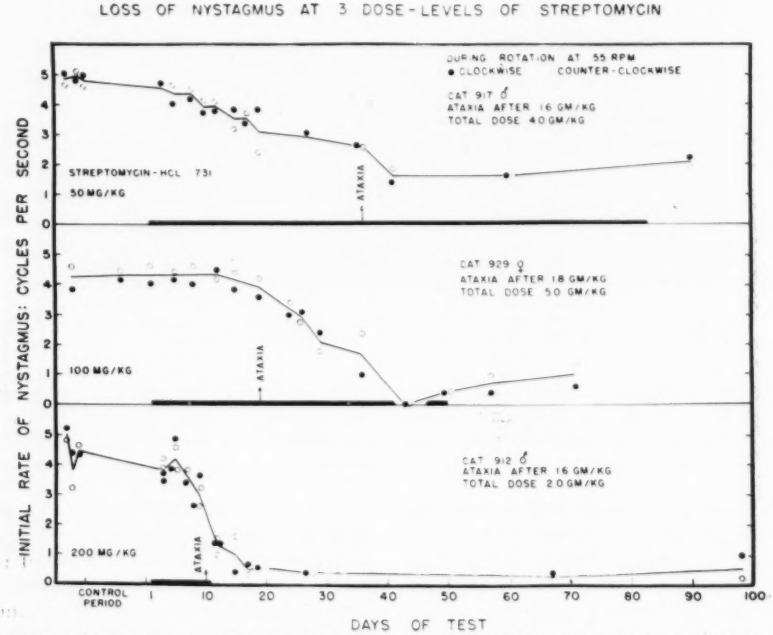

FIG. 2—Loss of nystagmus at three dose-levels of streptomycin. The black bars indicate the duration of treatment, the arrows the time of onset of ataxia.

vestibular response to rotation is lost, although some optokinetic response may persist (fig. 3). In some instances a partial recovery of the nystagmus has occurred after treatment was stopped.

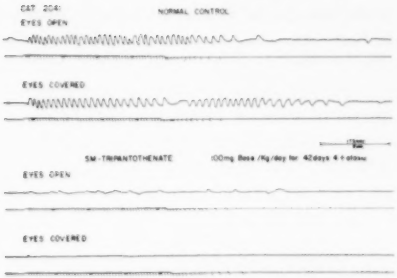

FIG. 3—Loss of optokinetic and vestibular nystagmus in a cat treated with streptomycin tripantothenate.

Even in animals in which the response to rotation has been completely lost, nystagmus has occurred in response to galvanic stimulation. This finding, first reported by Vanderhaeghe[50] in streptomycin-treated rabbits, is interpreted to mean that the central vestibular pathways remain intact even though the peripheral end organs have been damaged by the drug.

The Auditory Disturbance

Loss of hearing produced by streptomycin has been reported in relatively few instances. Most of these occurred in patients with tuberculous meningitis. At one time it was thought that the meningitis itself might be the cause of the deafness. It now seems more likely that streptomycin, which normally appears in the cerebrospinal fluid only in low concentration after intramuscular administration, reached the nervous system in much higher concentration in these cases, either because it was given by the intrathecal route, or because the inflamed meninges permitted more of it to enter the cerebrospinal fluid.

In cats, streptomycin has been shown to reduce the maximum voltage of the cochlear microphonic response when it is given by subcutaneous injection in doses of 200 mg. per kilogram daily or greater. No depression of the response has been seen in cats treated with 100 mg. per kilogram daily, even for 60 days or longer, unless a minute fraction (2 mg. per kilogram) of the dose is given intracisternally. In this case the microphonic response is severely depressed after only a few days of treatment (figs. 4 and 5).

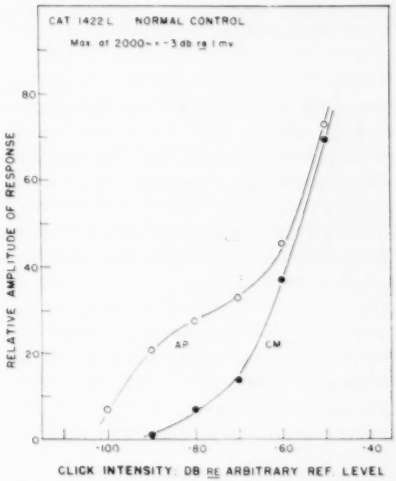

FIG. 4—Growth of the cochlear microphonic potential (CM) and auditory nerve action potential (AP) in a normal cat as a function of intensity of the click used as a stimulus.

Histologic Findings

Mushett and Martland[35] failed to find any lesions in the central nervous system of dogs which had shown the vestibular disturbance due to streptomycin, while Stevenson and his colleagues[47]

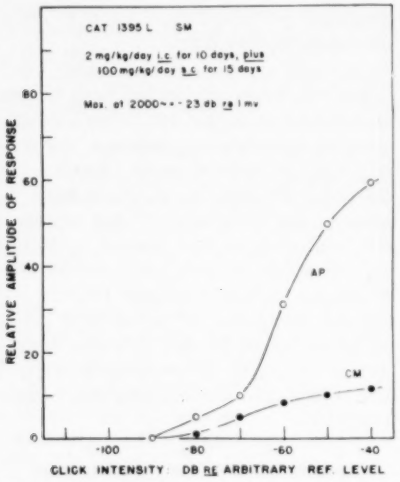

FIG. 5—Selective depression of the cochlear microphonic potential (CM) in a cat given streptomycin by both the intracisternal and the subcutaneous routes.

saw pathologic changes in the ventral cochlear nuclei (but not in the dorsal) of five patients who had become deaf while receiving streptomycin. Floberg and his co-workers[11] used a quantitative cytochemical method for measuring nucleoprotein in the cells of the vestibular ganglion and in Deiters' nucleus of the guinea pig and found substantial decreases after streptomycin treatment. Further evidence of a central vestibular lesion was obtained by Winston and his colleagues,[54] using the method of vital staining with trypan blue in streptomycin-treated cats.

A peripheral, end-organ lesion has been demonstrated by Caussé and his colleagues[6] in the mouse, by Berg[2] in the cat, by Rüedi and his colleagues[41] in the guinea pig, and by Hawkins and Lurie[19] also in the cat (fig. 6). Schmidt,[44] studying the brains of the cats treated by the last-named authors, found no significant changes in the cerebellum or in the brainstem nuclei. These divergent results are difficult to reconcile, but it should be noted that Rossi and his co-workers,[40] in a new series of papers, report nuclear, ganglionic and end-organ lesions in the guinea pig, and state that the end organs are affected only by large doses of the antibiotic. It appears that similar complete studies at several dose-levels will have to be made in various species before we can be certain whether the site of the damage is the same for all, or whether it varies from one species to another. The location of the lesion in man is particularly in need of clarification.

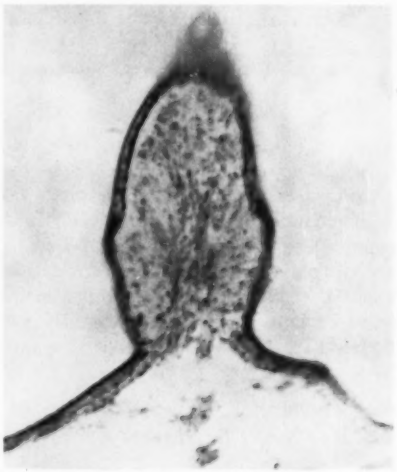

FIG. 6—Crista ampullaris in a cat treated with streptomycin, showing degeneration of the neuroepithelium.

DIHYDROSTREPTOMYCIN

Dihydrostreptomycin, the reduction product of streptomycin, was introduced into clinical use because it showed a significantly lower *vestibular* toxicity than streptomycin without any reduction in antimicrobial efficacy.[10,26,27] It also produced a lower incidence of minor

side effects, including sensitization reactions, and for a time it almost completely replaced streptomycin in the treatment of tuberculosis. From later clinical reports, however, it became clear that dihydrostreptomycin has a greater tendency than streptomycin to cause *impairment of hearing* in patients.[4,39,52] Profound deafness was reported by various European authors[3,51] in patients receiving dihydrostreptomycin intrathecally. Although the dihydrostreptomycin used in some of these clinical investigations appears to have contained impurities which made it more ototoxic than later, highly purified material, a large scale clinical comparison of samples of streptomycin and dihydrostreptomycin of known high purity has shown once more that prolonged treatment with dihydrostreptomycin is more likely to cause loss of hearing, just as streptomycin is more likely to cause vestibular disturbance.[8,32]

The incidence of vestibular and auditory disturbances in 195 tuberculous patients receiving streptomycin 1 gm. daily, and in 189 patients receiving dihydrostreptomycin 1 gm. daily for 120 days is shown in Table I. Table II shows the incidence of ototoxic reactions in two other groups of 82 and 84 patients, who received similar treatment with streptomycin and dihydrostreptomycin respectively. In 67 streptomycin patients and in 70 dihydrostreptomycin patients treatment was continued for 4 to 16 months beyond the original 120-day period.

The effect of dihydrostreptomycin is insidious, since the hearing loss may develop without warning, even after treatment has been stopped for days or even weeks.

In the cat, dihydrostreptomycin is about one half as toxic for the vestibular system as streptomycin. Its effect on the cochlear potentials of this species is peculiar, in that it actually enhances the microphonic response, while depressing the action potentials of the auditory nerve (fig. 7). This is exactly the reverse of the effect of streptomycin. Microscopic sections of the temporal bones from dihydrostreptomycin-treated cats show degeneration of the neuro-

TABLE I

OTOTOXICITY OF STREPTOMYCIN AND DIHYDROSTREPTOMYCIN

DRUG AND DOSAGE	REACTION			
	VESTIBULAR		AUDITORY	
	NO.	%	NO.	%
Rx 1 gm. per day for 120 days				
Streptomycin (82 patients)	25	30	2	2.5
Dihydrostreptomycin (84 patients)	9	11	12	14
Continued Rx 1 gm. per day for 4 to 16 months				
Streptomycin (67 patients)	6	9	12	18
Dihydrostreptomycin (70 patients)	6	8.5	20	28

Based on Mahady, et al.: Am. Rev. Tuberc., 68:238-248, 1953.

TABLE II

OTOTOXICITY OF STREPTOMYCIN AND DIHYDROSTREPTOMYCIN

	DURATION OF Rx (DAYS)	REACTION			
		VESTIBULAR		AUDITORY	
		NO.	%	NO.	%
Rx 1 gm. per day for 120 days					
Streptomycin (195 patients)	0-60	34		6	
	61-120	12		7	
(Post-Rx)	121-300	1		8	
		—		—	
	Total	47	24	21	11
Dihydrostreptomycin (189 patients)	0-60	7		8	
	61-120	8		16	
(Post-Rx)	121-300	1		36	
		—		—	
	Total	16	8.5	60	31

Based on Cohen, et al.: Am. Rev. Tuberc., 68:229-237, 1953.

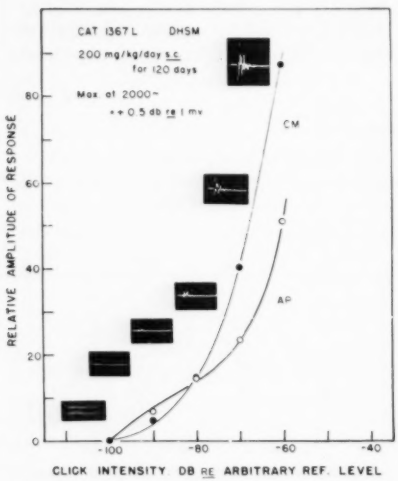

FIG. 7—Enhancement of the cochlear microphonic potential (CM) and depression of the auditory nerve action potential (AP) in a cat treated with dihydrostreptomycin.

epithelium of the vestibular end organs and of the external hair cells of the organ of Corti.

Since streptomycin shows its major toxicity for the vestibular system and dihydrostreptomycin for the auditory, the use of a 1:1 mixture of the two has been recommended in an attempt to minimize the effect of both antibiotics.[23,38] Although this simple but ingenious compromise has been shown to reduce the incidence of ototoxicity, it has been criticized on the basis that it does not necessarily avert the danger of delayed hearing loss due to dihydrostreptomycin.

HYDROXYSTREPTOMYCIN

Hydroxystreptomycin is an active antibiotic of the same group, as shown by its structural formula. Unfortunately, it

proved to be considerably more toxic for both the vestibular and the cochlear systems than either streptomycin or dihydrostreptomycin.[17] Because of its extreme ototoxicity observed in the cat, it was never released for clinical trial.

ately, the absorption of neomycin when applied locally or given by mouth is so low that it can be used freely in topical preparations and for sterilization of the gut. The pharmacology of neomycin has recently been reviewed.[18]

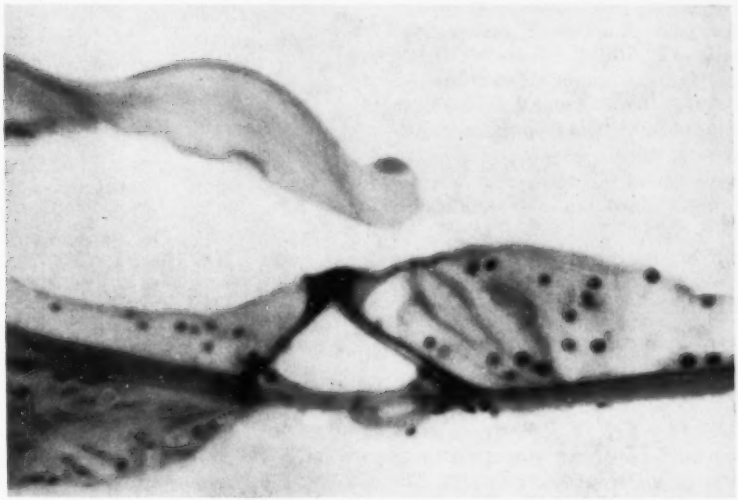

FIG. 8—Organ of Corti in a cat treated with neomycin, showing degeneration of the hair cells.

NEOMYCIN

Neomycin is another bad actor so far as the inner ear is concerned. Chemically it is related to streptomycin, but it exerts its ototoxic effect almost exclusively on the cochlear system. Profound hearing loss occurred in patients treated by Carr and his colleagues[5] and by Waisbren and Spink[53] with neomycin by the parenteral route. In laboratory animals it causes destruction of the organ of Corti (fig. 8) and other intracochlear structures.[20,42] It also has a serious nephrotoxic action, but the exact relationship between the action on the renal tubular epithelium and that on the organ of Corti has not been clarified. Fortun-

VIOMYCIN

Viomycin also shows both ototoxicity and nephrotoxicity, but they are apparently less serious than those of neomycin. In clinical use it has produced both cochlear and vestibular effects.[1] The chemical structure of viomycin has not yet been worked out in full, nor have any extensive studies been made in animals.

PANTOTHENATES

A number of reports have appeared in the literature describing the use of various substances to reduce the acute toxicity of streptomycin and dihydro-

streptomycin and to minimize their ototoxicity. The most promising of these has been pantothenic acid, introduced by Keller and his colleagues,[29] combined with the antibiotics in the form of tripantothenate salt. Impressive laboratory and clinical evidence to show its reduced toxicity has been brought forward,[9,14,30,37,46] but some German,[31] British[7] and American[22] investigators have found it difficult to substantiate the original German claims. Hewitt and his co-workers[24] have made a clinical comparison of dihydrostreptomycin-pantothenate and dihydrostreptomycin-sulfate and have found the ototoxicity low for both preparations but not significantly different. Katz and his co-workers[28] used 2 gm. of dihydrostreptomycin-pantothenate daily in tuberculous patients and found a substantial incidence of hearing loss.

GLUCURONATES

Rossi, Olivieri and Gay[40] have recently published a series of papers on streptomycin and neomycin glucuronates as preparations of reduced ototoxicity. The experimental documentation is impressive, since it includes extensive physiologic and histopathologic studies in guinea pigs. A report of the clinical evaluation of these salts is awaited with great interest. We may hope that it will include a direct comparison between streptomycin-sulfate and streptomycin-glucuronate in man.

KANAMYCIN

The latest addition to the family of basic antibiotics is the Japanese discovery, kanamycin.[48,49] Unfortunately it is also ototoxic, showing in lesser degree the same properties as neomycin, to which it is closely related in chemical structure.[12] Its vestibular toxicity is apparently less than that of viomycin but its cochlear toxicity is at least as great. Its usefulness in the therapy of tuberculosis is limited because of its tendency to cause hearing loss, but it may prove valuable and relatively innocuous in the short-term treatment of acute infections. Audiograms should be taken at weekly intervals if treatment is to last for more than ten days. In patients with preexisting impairment of renal function, kanamycin can cause severe, irreversible loss of hearing.

Table III represents an attempt to rank the five important basic antibiotics in terms of their vestibular, auditory and renal toxicity as exhibited in man and laboratory animals. Viomycin cannot be ranked with any assurance because so little information about its toxicity is available.

TABLE III

TOXICITY RATINGS OF BASIC ANTIBIOTICS

	VESTIBULAR	AUDITORY	RENAL
Streptomycin	+++	+	—
Dihydrostreptomycin	+	++	—
Viomycin	+?	+++?	++?
Neomycin	+	++++	++++
Kanamycin	+	+++	++

CONCLUSION

The existence of this number of antibiotics with ototoxic properties suggests that more will be forthcoming as the constant search for new chemotherapeutic agents goes on. If I might venture a suggestion in closing, you gentlemen, the Committee for the Conservation of Hearing, could perform a valuable service by calling the attention of your fellow physicians to the problem of the ototoxicity of these antibiotics and urging that they use these agents with appropriate care. Streptomycin and dihydrostreptomycin are the best understood members of the group. The newer agents should be approached with even greater caution. You might also wish to recommend that no new basic antibiotic be submitted for clinical trial without a thorough evaluation of its ototoxicity in at least two species of laboratory animals.

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DISCUSSION

GEORGE E. SHAMBAUGH, JR., M.D., Chicago: We are indebted to Dr. Hawkins for this very informative discussion of the ototoxicity of antibiotics. I would like to correct a statement he made in his last paragraph. He said that streptomycin and dihydrostreptomycin are the best understood members of the group. I think this is not correct. Dihydrostreptomycin is not well understood by the medical profession as an ototoxic drug. We are seeing a steady number of cases in our private practice of patients who received this drug, usually in combination with penicillin and sometimes also with streptomycin, who developed deafness three weeks to as long as six months afterwards.

The doctor who gave the drug usually has no idea that he has produced the deafness because he does not see the patient when the patient becomes deaf. The patient comes to the otologist. If we were not to inquire into the fact that the patient had been in a hospital in the previous six months, had had an operation, had received injections of a medication—possibly an antibiotic—and then if we were not to write to the hospital or to the doctor to find out what drugs the patient had received, we would never know that this patient's deafness was due to dihydrostreptomycin.

How do we know these patients lost their hearing because of dihydrostreptomycin? They are patients who had previously had perfectly normal hearing; they are young adults; they have no family history of deafness; and they have the rather abrupt onset for no apparent reason of a rather rapidly progressive neurosensory, perceptive, or nerve deafness, which comes on within a few days to several weeks after administration of the drug, reaches a low point, with perhaps a slight period of recovery, and then levels off at a permanent, irreversible and usually severely handicapping loss.

Now, we do not see this in any other condition. Young, healthy adults do not experience this type of deafness. With what we know about the delayed effect of dihydrostreptomycin on ototoxicity, we are correct, I believe, in concluding that these patients who have received dihydrostreptomycin some months before are deaf as a result of this drug.

This particular drug—dihydrostreptomycin—is extraordinary and unique among the ototoxic drugs. We all know that quinine, the salicylates, neomycin, streptomycin, and certain other of the newer antibiotics can cause deafness; but they cause it during or immediately after the administration of the drug.

I would like to ask Dr. Hawkins what it is that causes this latent period? It is a most extraordinary thing, and makes dihydrostreptomycin a particularly dangerous drug, because with quinine, the salicylates, streptomycin, and other ototoxic drugs the physician can stop the drug when the patients starts to show toxic reactions, whereas with dihydrostreptomycin, "the horse is long since gone" by the time the deafness appears and there is no way to withdraw the drug given months before.

We have now seen some thirty-odd cases of deafness in patients from dihydrostreptomycin. Usually it has been given in combination with penicillin in comparatively small doses as a prophylactic following an operation. In a few cases it was given for severe infections, but streptomycin could have been given just as well and the patient today would not be deaf.

One of these patients has, at the present time, a loss of 50 to 60 decibels. He has a speech discrimination score in one ear of eight per cent, and in the other ear of 18 per cent. In other words, he is practically unable to comprehend speech, either by the spoken voice or by the amplified voice. This is a very serious handicap indeed.

As a member of the Committee on Conservation of Hearing, I have circularized the other members of the Committee with regard to this problem to see how many others were seeing such cases. I was rather surprised that only a few members of this Committee had observed such cases. The reason may be that in all cases of nerve deafness, they have not asked for specific information regarding previous operations, hospitalization, or illnesses for which an antibiotic containing dihydrostreptomycin may have been given. Dr. Howard House has seen a number of these cases, and we are including them in a report we expect to publish.

To summarize, dihydrostreptomycin is a very dangerous ototoxic drug, dangerous, first, because of the delay of weeks or months before the onset of the hearing loss and, second, because in susceptible individuals—and there must be a great difference in individual susceptibility—as little as a total of 1.5 or 2.0 grams of this drug can cause irreversible permanent handicapping nerve deafness. Its danger is increased because it is used by many of the drug houses—perhaps all of them—in their combinations of antibiotics which usually contain streptomycin, dihydrostreptomycin and penicillin with nothing on the label to indicate that this, the very dangerous ototoxic dihydrostreptomycin, is present in the medication.

To my mind, dihydrostreptomycin should not be used parenterally at all. Streptomycin will accomplish exactly the same thing for the gram-negative organisms; and, if there is a toxic effect on the vestibular mechanism, it becomes immediately apparent and the drug can be diminished or withdrawn.

WALTER E. HECK, M.D., San Francisco: In the past week I have had occasion to review some audiograms of patients with tuberculosis treated with kanamycin. There were about a dozen cases; in three of them there were definite hearing losses attributable to the drug.

The point I wish to emphasize is that the quantity of drug used in these cases was even less than was cited by Dr. Hawkins, indicating that this is a very ototoxic drug.

I have been very distressed in the past by the hearing losses induced by neomycin. As you know, it is usually used as heroic therapy in a case where nothing else is effective, and the patient might die if it is not used.

At a recent meeting of the San Francisco branch of the American Federation for Clinical Research it was pointed out that apparently the strains of bacteria which are drug-sensitive, and those which are resistant, are identical for neomycin and kanamycin. Thus there appears to be no reason for allowing ourselves to be rushed into using kanamycin, when a need for heroic therapy arises, just because it is a new drug.

A ZONULE STRIPPER

ALSTON CALLAHAN, M.D.
BIRMINGHAM, ALABAMA

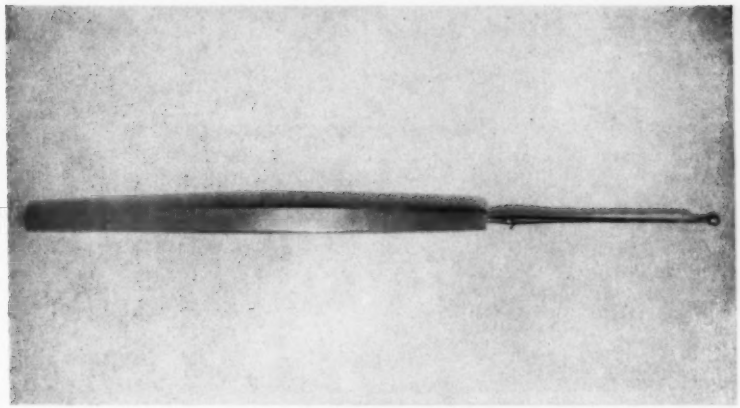

FIG. 1—Zonule stripper.

THE shaft of the zonule stripper is similar to that of a muscle hook, with a curve near the end conforming to the anterior lens surface, and a flattened, round, symmetrical tip (figs. 1 and 2). After the section has been completed and the indicated surgical operation on the iris performed, the tip is introduced through the section and pupillary aperture, and slid along the anterior surface of the lens to the 3 o'clock position at the limbus (fig. 3, A). The tip must not be permitted to advance so far that the hyaloid membrane will be ruptured or the vitreous dislodged, but it must be inserted far enough to reach the fibers of the zonular ligament attached to the anterior capsule (fig. 3, B). The tip is then moved in a clockwise direction, from the 3 o'clock position to the 9 o'clock position. Unless unusual resistance from the fibers is encountered, it is necessary to pass the tip around only once. Then only the fibers which insert into the equator and posterior capsule remain attached, so that the force necessary to pull the lens away from the remaining fibers by means of the erysophake or capsule forceps is greatly reduced.

The lower equator of the lens may then be tumbled up from below and the lens removed. Or if desired, this stripper or a regular muscle hook can be used to strip away the fibers above, permitting the lens to be slid out.

Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

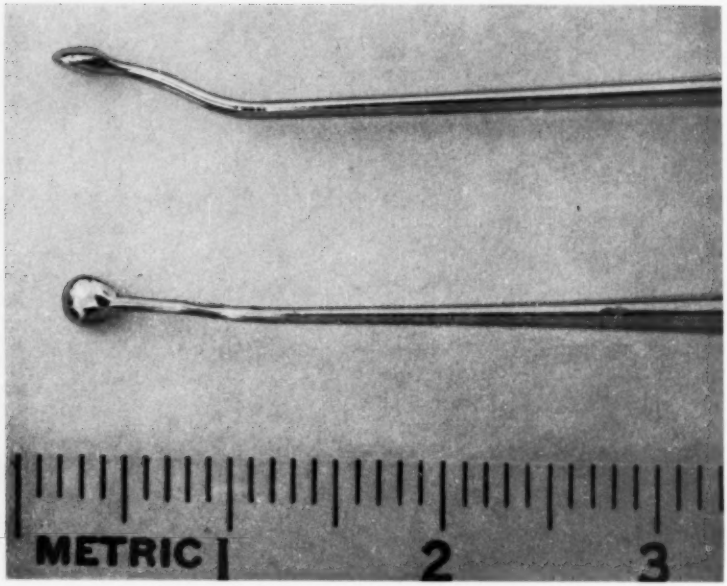

FIG. 2—Enlarged view of end and tip of zonule stripper.

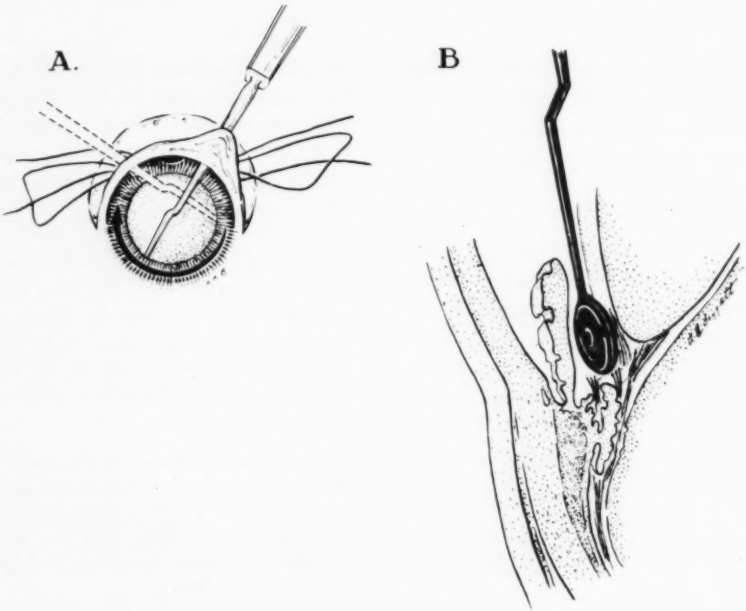

FIG. 3—Use of the zonule stripper.

The dangers of tearing away the fibers without looking at them have been described in the literature, but we have found that the maneuver of stripping away the anterior fibers without disturbing the vitreous can be done with reasonable care, which saves the eye the much greater trauma of trying to break the fibers by pressing on the outside of the eye. The idea of a zonule stripper or a "zonulotome" is not new: de Andrade devised several models in 1948, but these instruments were not widely adopted. With the use of Xylocaine and hyaluronidase retrobulbarly, the eye can be so softened that these methods should be reappraised. Actually there is more hazard for most ophthalmic surgeons in Kirby's method of direct stripping because of the danger of rupturing the capsule or loss of vitreous from elevation of the lens.

The chief indication for the use of the stripper is in extraction of cataracts from young adults whose zonular ligament is quite strong. The second indication is in those extractions in which the vitreous flows into the anterior chamber after the completion of an uncomplicated corneal section. In this complication, external pressure against the limbus for rupture of the fibers is contraindicated because vitreous may enter the wound if a modified Smith method or the Haik maneuver is used. In all of my intracapsular cataract extractions, the stripper is used.

BALL-POINT EYE SCISSORS

JOHN HARRY KING, JR., M.D.
WASHINGTON, D. C.

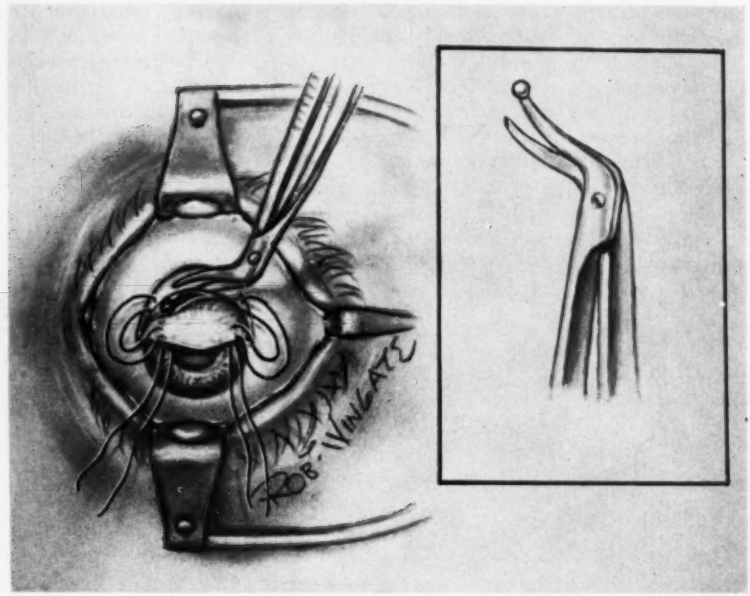

FIG. 1—Ball-point scissors pushing iris forward. (Insert) Close-up of scissors showing ball point.

THE iris may be inadvertently incised when corneal scissors are used to enlarge the corneoscleral section in cataract surgery or when the host cornea is being removed during penetrating keratoplasty. An accidental iridotomy is especially prone to occur when the points of the scissors cannot be observed deep in the anterior chamber angle or when the cornea is opaque. Cataract scissors and corneal scissors have been designed* with a small ball protruding from the lower blade and extending forward from the cutting blades. This pushes the iris away from the advancing scissors and prevents its incarceration between the blades.

From the Eye Research Sections of Georgetown University Medical Center, and the Washington Hospital Center, Washington, D. C.

This research was supported, in part, by a research grant, B-975 (R), from the National Institute of Neurological Diseases and Blindness, National Institutes of Health, Public Health Service. It was also financed by the Washington Eye Bank (Lions 22-C). Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

*Manufactured by Storz Instrument Co., St. Louis.

A MUSCLE HEADLIGHT

HOBART A. LERNER, M.D.
ROCHESTER, NEW YORK

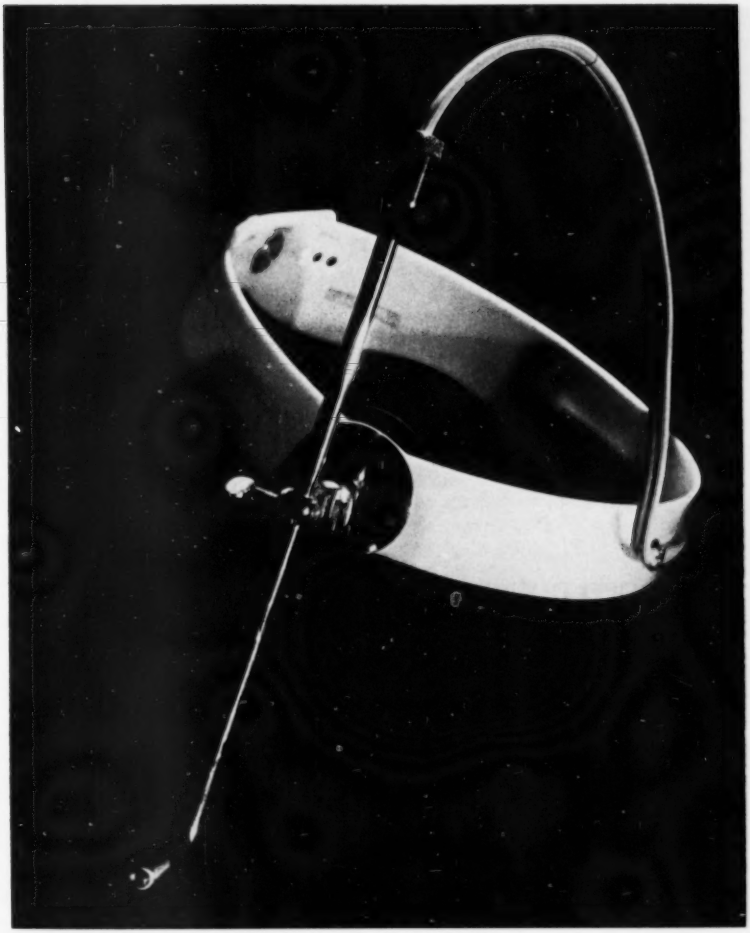

FIG. 1—Lerner muscle headlight.

THIS headlight consists of a plastic head-band with an adjustable long angulated Finoff transilluminator stem pro-

Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

vided with a 2.5 volt Welch Allyn bulb. The stem with the bulb is adjustable up and down. It can also be moved from side to side and forward or backward; in fact, the bulb can be adjusted to any angle or any position. I generally wear it so that the bulb extends down and out from the position of my nose. The transilluminator is wired so that I can attach it to the transformer on my unit, and it is also attached to a foot switch so that I can turn the bulb on and off merely by toe action on the foot switch.

I have found that this apparatus is invaluable in muscle study. I am able to use both hands for the prism cover test and for positioning the patient's head. When examining a small child, I can attract his attention by flicking the foot switch and turning the light on and off relatively quietly. I can move my head from side to side to move the position of the bulb, thus attracting the child's attention further. I am able to see the Hirschberg reflexes.

This piece of equipment was constructed specifically for me by Welch Allyn but is not yet in general production.

THE GUARDED DOUBLE-BLADED SCLERAL RESECTION KNIFE

SEYMOUR B. GOSTIN, M.D.
DALLAS, TEXAS

RETINAL detachment is being treated more and more by some form of lamellar scleral resection. The most difficult portion of this operation is the outlining of the lamellar flap by parallel incisions of

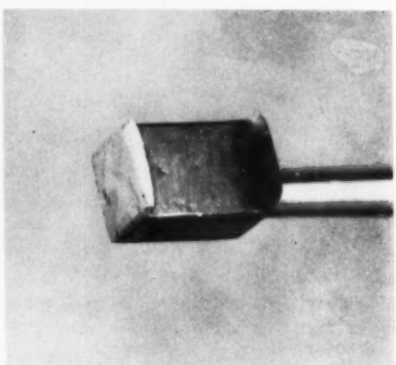

FIG. 1—The working portion of the instrument.

Chief, Ophthalmology, Veterans Administration Hospital, and Assistant Professor of Ophthalmology, Southwestern Medical School of the University of Texas, Dallas, Texas.

The statements of the author do not necessarily reflect the opinions of the Veterans Administration.

Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

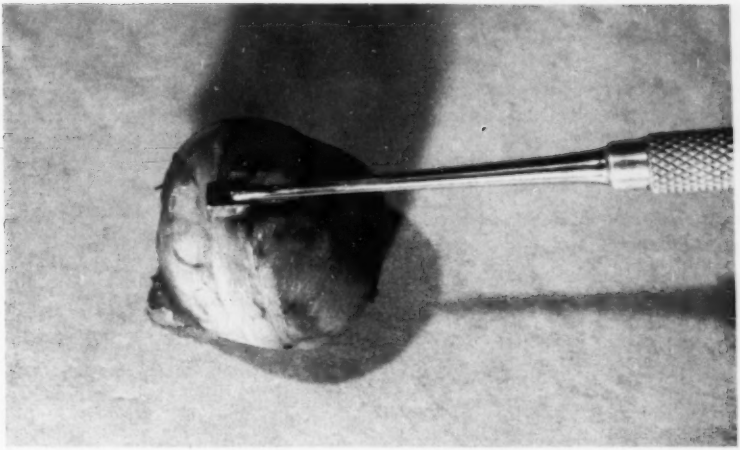

FIG. 2—Parallel incisions made on enucleated eye.

proper depth in the sclera. Much of the difficulty encountered stems from the poor exposure available for procedures to and beyond the equator of the globe.

The guarded double-bladed scleral resection knife* was designed to make two parallel incisions of predetermined depth simultaneously in the sclera. It can do this even in the posterior areas of the globe where exposure and working room are all but lacking, permitting this task to be accomplished in a matter of a few minutes. The resulting incisions are perpendicular to the surface of the globe thus facilitating the emplacement of sutures.

*Manufactured by Storz Instrument Co., St. Louis.

PHOTO AUDIO CONDITIONING MACHINE FOR VERY YOUNG CHILDREN

E. M. Herzon, M.D.
ELGIN, ILLINOIS

With the advent of a commercially available automatic motor-operated, magazine-loaded, 35 mm. slide projector,* it suddenly became feasible to make an economically useful adaptation for audiographic testing of very young children quite rapidly, as if an adult audiogram were being made. It is not necessary to open up the electrical connections of your present audiometer. The tone interrupter is synchronized to the slide changer with a normally "on" plunger-type switch.

Although a full discussion of the technique and psychological conditioning involved will be the subject of another paper, in brief, the automatic changing picture on a screen is made the "reward" for acoustic conditioning which can be obtained in 10 to 15 seconds. The audiogram is then obtained in a manner much like that used in ordinary audiometry with the use of a push button to signify the reception of a tone, thus changing a 35 mm. slide in the projector instead of making a red light go on in the audiometer, a technique with which you are all familiar.

It is very important that all extraneous visual and auditory impulses be minimized while using this conditioning technique, otherwise the result will not be any better than current attempts at audiometry with a young child.

The child is led into a well lighted, cheerful, soundproof room. He is comfortably seated in a chair, and the headphones are put on. The push button is placed in the child's hands. The only other thing in the room is a small 24 inch screen upon which the pictures are projected through the soundproof window. Thus all the noisy apparatus, such as slide changer, projector, audiometer and tester, are outside of the room (fig. 1). An assistant may remain in the room with the child to help establish the proper conditioning response by nudging the child's finger the first few times a tone is presented. After this, usually no more help is needed. Both the ear-

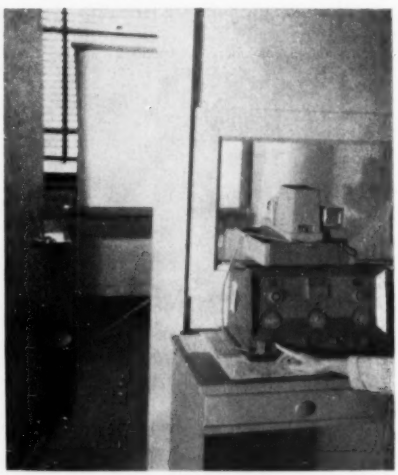

FIG. 1—Automatic slide projector rests on top of chassis of audiometer and shoots through triple glass window into soundproof room. Picture screen visible through doorway. Finger points to electrical switch.

Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

*Argus, Kodak, Revere.

phone cord and the push-button cord are led into the soundproof room through a hole in the wall, which is then plugged with cotton. By means of an inside mirror the tester can be comfortably seated on the outside and still watch the button in the hand of the child for hand movement tip-offs to the auditory tones as presented.

Several 35 mm. slide projectors are now on the market which come with optional attachment of the same motor-operated magazine-loaded slide changer. A two-pronged electrical plug from the attachment simply plugs into an outlet on the projection machine. Instead of this an ordinary triple socket plug is inserted here and a lead is taken off "in series" to a normally "on" plunger-type switch (current rating: 3 amp., 125 volts), which is mounted in proximity to the tone interrupter on the front panel of the audiometer as shown (fig. 2). Since the plunger switch is normally in an "on" position, this is actually the position when the circuit is complete and pressure on the push button will change the slide. Then when the tone interrupter arm is moved to a *vertical contact position* the plunger of the switch is pushed inwards, thus putting the switch in an "off" position which breaks the circuit and makes it impossible for the slide changer to be actuated by the push button. The contact position of the tone interrupter is adjusted so that the tone synchronously goes off with the switch in the "off" position.

In effect then, the adjustment is made so that the child is unable to make changes in the slides as long as the tone is absent; but when the tone is present, pressure on the push button will result in the slide always changing. Therein lies the "reward" part of the conditioning.

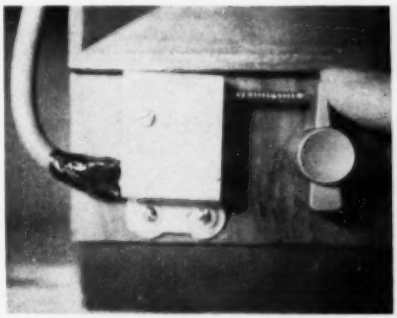

FIG. 2—Electrical switch with spring plunger which contacts the tone interrupter of audiometer.

With this machine over 500 audiograms have been obtained since September 1957. This experience allows me to conclude that the audiograms so obtained have the same diagnostic accuracy as regular audiograms, notwithstanding the age of the child. Furthermore, serial audiograms can be obtained with complete confidence as to reliability, thus proving to be a very valuable tool in judging the effectiveness of therapy.

OPERATING MICROSCOPE OBSERVATION VIEWER AND MOTION PICTURE CAMERA

WILLIAM F. HOUSE, M.D.
HOWARD P. HOUSE, M.D.
JACK URBAN
LOS ANGELES, CALIFORNIA

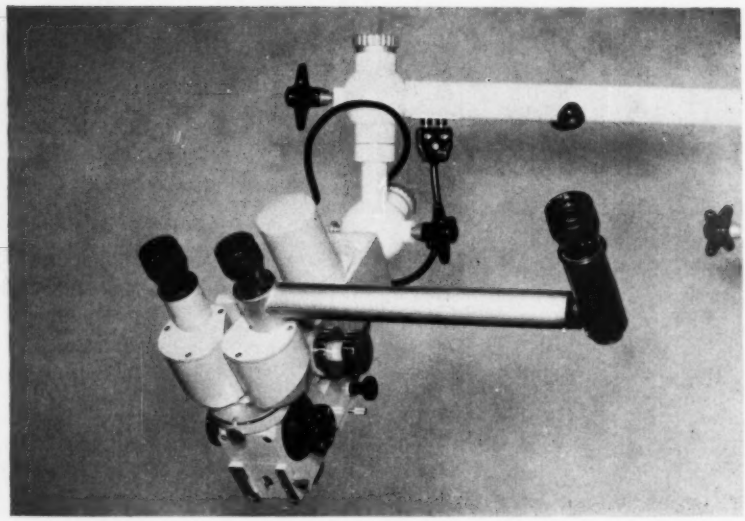

FIG. 1—The viewer in place on the microscope.

IN modern ear surgery the use of the operating microscope has become essential. There has, therefore, been considerable interest in recent years in developing a device to allow an observer and the surgeon to view the surgical field simultaneously.

Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

MICROSCOPE VIEWER

Several microscope viewers have been constructed. Dr. John Shea and the Carl Zeiss Company have these instruments available.

There are several important features of such a viewer. It should, in our opinion, have the same magnification, field size, and orientation as the surgeon's view. In addition, it is desirable to be able to move the viewer to either side of the microscope so that the student can be

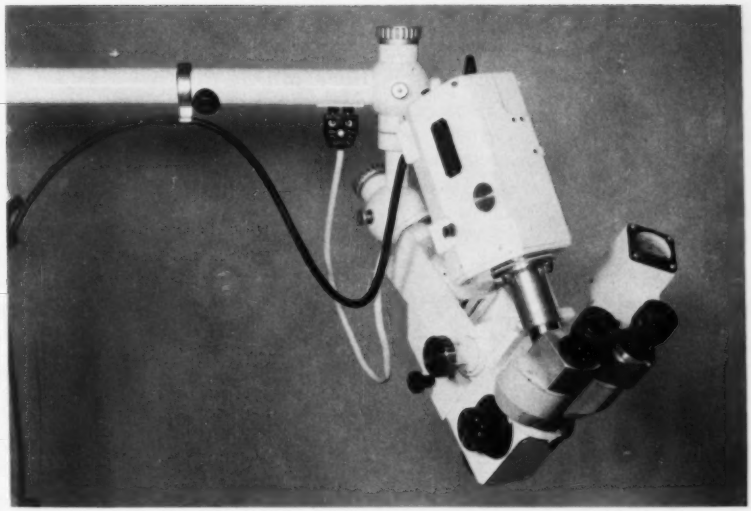

FIG. 2—The 16 mm. camera in place on the microscope.

placed at the head of the table regardless of the ear being operated upon.

MOTION PICTURE CAMERA ALLOWING SIMULTANEOUS PHOTOGRAPHY AND VIEWING THROUGH THE OPERATING MICROSCOPE

We have also developed a motion picture camera for the microscope. This camera is designed so that it covers a field size of approximately 17 mm. at the sixth power, and a 6 mm. field size at the sixteenth power. This allows the picture to fill the screen from top to bottom through a 6 mm. speculum at the sixteenth power. The 6 mm. speculum is the one conventionally used in most stapes mobilization surgery.

A very important feature of this camera is that the image should be oriented to the surgeon's viewpoint. Satisfactory pictures can be taken using Anscochrome film; however, we feel that better pictures will be obtained using Super Anscochrome, which is available on special order.

The camera used is a conventional motor-driven gun camera. On the right ocular an exposure meter is mounted to allow a constant check on the amount of light entering the microscope.

A camera has also been developed by Dr. John Shea, and we understand that the Carl Zeiss Company is also currently developing such an instrument. In addition to this type of camera, an oto-camera has been developed by Mr. Brubaker and Drs. Shambaugh and Derlacki. This camera does not use the microscope lens system and, therefore, has a number of advantages and some disadvantages over the above type of system.

The camera and the viewer are both available in models for either the straight ocular or the angled ocular.*

*Manufactured by the Urban Engineering Co., 6717 Melrose Ave., Los Angeles.

A NEW LARYNGOSCOPE BLADE

EUGENE S. HOPP, M.D.
SAN FRANCISCO, CALIFORNIA

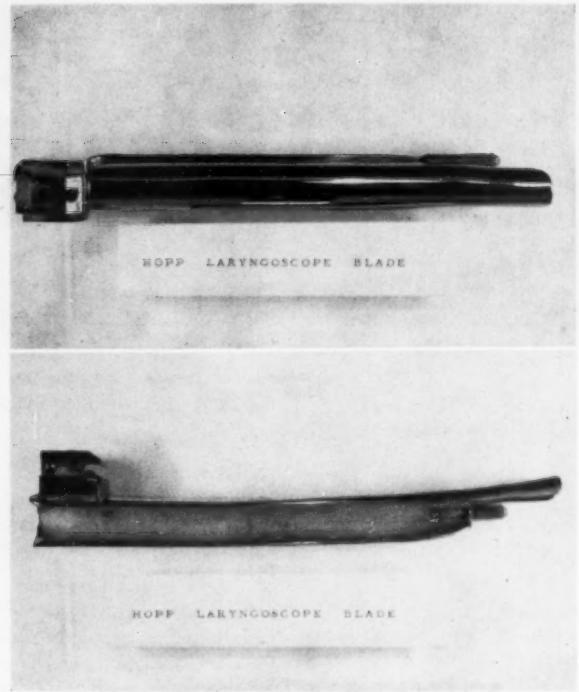

FIG. 1—Two views of Hopp laryngoscope blade.

THE introduction of a new laryngoscope blade necessitates careful and prolonged consideration in view of the many excellent ones presently available. After much study, it was felt that it might be possible to devise a new blade which would combine the best and most essential properties of its many predecessors (fig. 1).*

An open-sided blade was selected for two reasons. It permits a better visualization of the larynx, since instruments

From the Division of Otorhinolaryngology, University of California School of Medicine, San Francisco, and Hahnemann Hospital, San Francisco.

Presented as a *New Instrument* at the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1958, Chicago.

*Manufactured by V. Mueller and Co., Chicago 12.

do not have to be manipulated through the laryngoscope and so partly obstruct the field of vision. It enables an endotracheal tube to be inserted, should this prove necessary during general anesthesia, without the difficult problem of removing a closed laryngoscope while maintaining a tube in the larynx.

The side opening of the blade was widened, as the only essential function of the lower portion of the half-circle is to keep the tongue to one side of the operative field.

The spatulated distal end of the standard blade, which precluded its use inside the larynx, was replaced by an ovoid shaped distal end. This resembles several of the anterior commissure laryngoscopes. It enables one to insert the blade into the larynx and even between the cords, when necessary, without undue trauma.

The third and probably most important change is the upward inclination of the distal third of the blade. This provides those few added millimeters of exposure at the anterior commissure so essential in a difficult case.

The progenitor of this blade has been in constant use five or more years. It has proved most satisfactory in exploratory and operative laryngoscopy. Other otolaryngologists in the San Francisco area, impressed by its advantages, have obtained hand wrought duplicates. Anesthesiologists, too, have found this laryngoscope helpful in endotracheal intubation.

AN IMPROVED NASAL SPECIMEN COLLECTOR

EDLEY H. JONES, M.D.
VICKSBURG, MISSISSIPPI

THE easiest and most satisfactory way to collect nasal and/or sinus discharge for cytologic and bacteriologic study, is to use a sterile specimen collector.

Several specimen collectors have been devised, all good for certain purposes, but in my experience they have not been entirely satisfactory in difficult cases, too short in some patients and unable to enter the middle meatus in others. In patients with curved nasal passages, a collector with a straight tip could not be used.

For these cases I have designed a modification of Davison's 9 cm. specimen collector,* increasing the length to 10 cm., slightly curving the tip and flattening it vertically so that it can be directed past an enlarged turbinate or a curved septum into the posterior naris and the middle meatus (fig. 1). I have mounted the metal tube with a screw base so that the curved tip may be turned left or right, as desired. This assembly is

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*I wish to thank Mr. Henry Pilling for his cooperation in perfecting this Collector.

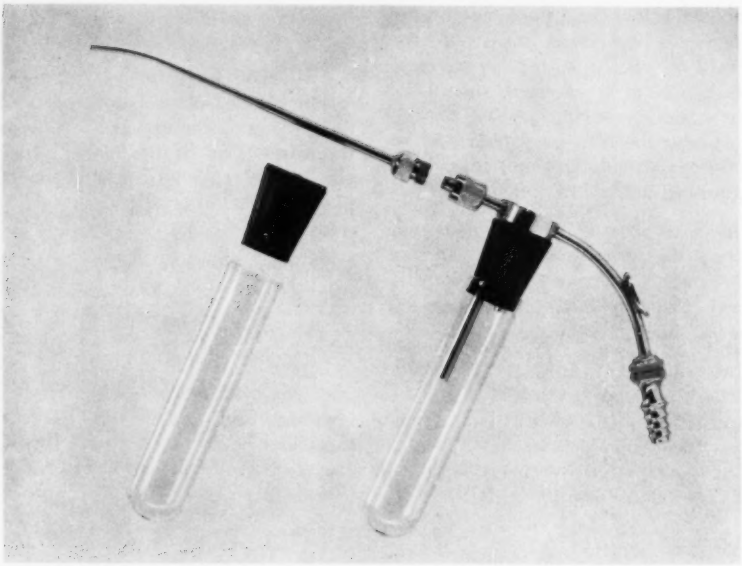

FIGURE 1.

mounted in a rubber stopper, fitting into a small test tube. After collecting the specimen, the glass tube can be removed, a sterile rubber stopper inserted, and the specimen sent to the laboratory. Extra test tubes and rubber stoppers are obtainable, and the units can be sterilized and kept in readiness for use.

I have found this specimen collector satisfactory in the difficult case as well as in the routine, or usual, case.

TRANSACTIONS

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PLEOPTICS

During the past few years the interest in the problem of strabismic amblyopia has become both wider and deeper. By various, in part rather elaborate, measures, investigations into the nature of amblyopia have been carried out and attention has turned to a number of the unusual features of this most interesting form of functional reduction in visual acuity. The fixation pattern of amblyopic eyes has been studied by subjective procedures (afterimages) and objective procedures (electro-ophthalmography). The puzzling fact that an amblyopic eye may have a rather high visual acuity when tested with isolated symbols, whereas the vision is considerably reduced when the symbols are presented in a row, is as yet not fully explained. The cases of amblyopia which are most difficult to handle, those with eccentric fixation, have undergone intensive study, particularly in some European centers.

The great amount of work expended on the solution of the amblyopia problem can not fail to result eventually in an improved understanding of the condition and in an improvement of our therapeutic procedures. It behooves both the ophthalmologists and the orthoptists to become acquainted with the new thoughts and methods in this field, and the American Orthoptic Council has therefore decided that the next Joint Meeting of the American Orthoptic Council and of the Association of American Orthoptic Technicians in October 1959, to be held as is customary in conjunction with the meeting of the American Academy of Ophthalmology and Otolaryngology, shall be devoted to a symposium on pleoptics. It is to be expected that the presentations will prove of considerable interest to everyone, both in regard to the theory of amblyopia and to the first results obtained with the new forms of treatment, originated in Europe, as applied to the conditions prevailing in the United States.

HERMANN M. BURIAN, M.D.

BASIC COURSE IN ORTHOPTICS TO BE CONTINUED

During the summer of 1958 the basic course in orthoptics sponsored by the American Orthoptic Council was given for the first time in Ann Arbor. Prior locations within recent years had been at Washington University in St. Louis, and at the University of Iowa. It was possible to organize the course in 1958 as a regular summer session course of the University of Michigan, allowing the registrants the full advantages of student enrollment in the University. This included facilities of the Student Health Service, the Women's League, and the women's athletic facilities, including the use of the swimming pool. Individual counselling, as necessary, was carried out by representatives of the office of the Dean of Women. Housing was arranged in dormitory facilities adjacent to University Hospital, where the lectures were held. Twenty students were registered for the eight week session, and all completed the course. Many of them are continuing with their ten-month practical training at the present time, and will be qualified for their examination in orthoptics for certification by the Council in 1959.

A large percentage of the class had more than the minimum educational background required by the Council for admission, and the lecturers were unanimous in their reaction that the task of teaching was lightened considerably by this factor. It appears evident that a more rigid adherence to preliminary education will attract better candidates for orthoptics, and better information must be placed in the hands of vocational advisers in order to continue an adequate flow of students. During the past year orthoptic training has been listed in several fundamental publications for vocational counsellors, and the number of inquiries for the Council brochure has been increasing steadily.

The basic problem of the type of course to be given still has not been solved, however, since there are generally two groups of students taking the basic instruction. The first are those to whom orthoptics is an entirely new subject, and for these much preliminary teaching of nomenclature and basic concepts is necessary. The second group comprises those students who have already begun orthoptics in some center, and who come to the summer course to study the basic material more thoroughly. With the number of registrants taking the course at present, it is impossible to present two separate courses; but if growth in numbers of students occurs in the future, the Council should consider seriously the possibility of presenting both a fundamental course for beginners and a second one for those with a greater background.

Plans are now under way for presentation of the Basic Course in Orthoptics to be held at the University of Michigan during the summer session from June 22 through August 15. The lectures will be held at the University Hospital, housing will be provided in the University dormitories, and practical demonstrations will be arranged again utilizing facilities in Detroit.

For further information with regard to the course, please contact John W. Henderson, M.D., Room 6155, Outpatient Building, University Medical Center, Ann Arbor, Michigan.

JOHN W. HENDERSON, M.D.

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November OPTICS AND PRINCIPLES OF BIOMICROSCOPY	Bourne Jerome Charles J. Campbell
December REFRACTION	Manuel L. Stillerman Bernard C. Gettes
January OCULAR MICRO-BIOLOGY, EXTERNAL DISEASES, AND ASEPTIC TECHNIQUE	Henry F. Allen Samuel J. Kimura
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June OTOLOGICAL ACOUSTICS	Donald H. Eldredge Juergen Tonndorf

CONSULTANTS:

H. C. Ballenger, L. W. Dean, Jr., J. Brown Farrior, A. C. Hilding, Arthur L. Juers, Clair M. Kos Clifford F. Lake, John R. Lindsay, Moses H. Lurie, Carl H. McCaskey, William J. McNally, Werner Mueller, Jack E. Mulsow, Robert E. Priest, Scott N. Reger, Herman Semenov, Ben H. Senturia, G. E. Shambaugh, Jr., Maurice F. Snitman, William C. Thornell, O. E. Van Alyea, Theodore E. Walsh, Henry L. Williams.

The Courses begin on September 1 each year and run continuously for ten months.

Application blanks and information may be secured from the Executive Secretary-Treasurer of the Academy: Dr. William L. Benedict, 15 Second Street S.W., Rochester, Minnesota.

REPORT OF THE COMMITTEE ON CONSERVATION OF HEARING

THE following "Guide for the Evaluation of Hearing Impairment," prepared by the Subcommittee on Noise, has been adopted for publication by the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology.

DEAN M. LIERLE, M.D.
Chairman, Committee on Conservation of Hearing

Guide for the Evaluation of Hearing Impairment

The following statement of principles, prepared by the Subcommittee on Noise and approved by the Council of the American Academy of Ophthalmology and Otolaryngology, is presented as a guide for the evaluation of hearing impairment* regardless of the cause or causes of such impairment. These principles are based on current medical opinion. The Subcommittee on Noise reaffirms the principles concerning the evaluation of hearing impairment already set forth jointly by it and by a cooperating subcommittee of the American Medical Association.** The Subcommittee on Noise now recommends the following interim method for measurement and calculation.

Ideally, hearing impairment should be evaluated in terms of ability to hear everyday speech under everyday conditions; the term "impairment" will be used hereinafter in this sense only. The ability to hear sentences and repeat them correctly in a quiet environment is taken as satisfactory evidence of correct hearing for everyday speech. Because of present limitations of speech audiometry, the hearing level*** for speech should be estimated from measurements made with a pure tone audiometer. For this estimate, the Subcommittee recommends the simple *average* of the hearing levels at the three frequencies 500, 1000 and 2000 cps.

In order to evaluate the hearing impairment, it must be recognized that the range of impairment is not nearly as

*"Impairment" is here used as defined in "Guides to the Evaluation of Permanent Impairment," approved by the Committee on Medical Rating of Physical Impairment, Journal of the American Medical Association, September 27, 1958, Volume 168, Number 4, page 475. "Impairment," as defined by the American Medical Association, denotes a medical condition that affects one's personal efficiency in the activities of daily living. It is recognized that "disability," as used in various workmen's compensation laws, involves nonmedical factors, since it may be related to actual or presumed reduction in ability to remain employed at full wages. Permanent impairment is, therefore, a contributing factor to, but not necessarily an indication of, the extent of a patient's permanent disability within the meaning of the workmen's compensation laws.

**Report of the Council on Physical Medicine & Rehabilitation, Journal of the American Medical Association, April 1955, Volume 157, pages 1408-1409, "Principles for Evaluating Hearing Loss."

***The hearing level of the listener is the number of decibels that his threshold of hearing lies above the standard audiometric zero for that frequency. It is the reading on the so-called "hearing loss" dial of an audiometer that is calibrated according to American Standard Z 24, 5-1951 (American Standards Association, March 21, 1951).

wide as the audiometric range of human hearing. Audiometric zero, which is presumably the average normal hearing threshold level, is not the point at which impairment begins. If the average hearing level at 500, 1000, and 2000 cps is 15 db or less, usually no impairment exists in the ability to hear everyday speech under everyday conditions. At the other extreme, however, if the average hearing level at 500, 1000 or 2000 cps is over 82 db, the impairment for hearing everyday speech should be considered total. The Subcommittee on Noise recommends the following formula: for every decibel that the estimated hearing level for speech exceeds 15 db, allow 1.5 per cent up to the maximum of 100 per cent. This maximum is reached at 82 db.

At this time, the Subcommittee on Noise makes no specific recommendation regarding a correction for a shift in the hearing threshold due to age, because the relation of presbycusis to noise-induced hearing loss is not yet fully understood.****

The Subcommittee on Noise recommends that any method for the evaluation of impairment include an appropriate formula for binaural hearing which will be based on the hearing levels in each ear tested separately. Specifically, the Subcommittee on Noise recommends the following formula: the percentage of impairment in the better ear is multiplied by five (5). The resulting figure is added to the percentage of impairment in the poorer ear, and the sum is divided by six (6). The final percentage represents the binaural evaluation of hearing impairment.

****For current information about the relation of hearing threshold to age, see Table XXII, 1954 Wisconsin State Fair Hearing Survey (Median Values for Office Workers). Published by the American Academy of Ophthalmology and Otolaryngology, 1957.

Examples illustrating the use of the above recommendations:

cps	500	1000	2000	3000	4000	6000
Rt. (db)	30	35	45	55	60	50
Lt. (db)	35	40	55	60	70	65

Frequencies most important for the understanding of speech.

Right Ear (db)	*c/s*	*Left Ear* (db)
30	500	35
35	1000	40
45	2000	55
110		130

$\frac{110}{3}$ db = 36⅔ db estimated hearing level for speech

$\frac{130}{3}$ db = 43⅓ db estimated hearing level for speech

To convert estimated hearing level for speech to per cent impairment of hearing:

Right Ear:

36⅔ db estimated hearing level for speech
−15 db hearing level for speech below which no impairment of hearing for speech usually exists.
21⅔ db rounded out to nearest digit = 22 db
22 db × 1.5% per decibel = 33% impairment of hearing in right ear.

Left Ear:

43⅓ db estimated hearing level for speech
−15 db hearing level for speech below which no impairment of hearing for speech usually exists.
28⅓ db rounded out to nearest digit = 28 db
28 db × 1.5% per decibel = 42% impairment of hearing in left ear.

To convert the monaural impairments to per cent binaural impairment the formula is:
[(per cent impairment in better ear × 5) plus (per cent impairment in worse ear)] ÷ 6.

Therefore
[(33% × 5 = 165%) + (42% × 1 = 42%)] ÷ 6 = 34.5% binaural impairment.

cps	500	1000	2000	3000	4000	6000
Rt. (db)	50	60	75	85	90	80
Lt. (db)	10	10	20	35	50	45

Frequencies most important for the understanding of speech.

Right Ear (db)	*c/s*	*Left Ear* (db)
50	500	10
60	1000	10
75	2000	20
185		40

$\frac{185}{3}$ db = 61⅓ db estimated hearing level for speech

$\frac{40}{3}$ db = 13⅓ db estimated hearing level for speech

To convert estimated hearing level for speech to per cent impairment of hearing:
61⅓ db estimated hearing level for speech
−15 db hearing level for speech below which no impairment of hearing for speech usually exists.
46⅓ db rounded out to nearest digit = 47 db
47 db × 1.5% per decibel = 70.5% impairment of hearing in right ear.

Go through the calculation again for left ear. The per cent impairment of hearing is zero in this case.

To convert the monaural impairments to per cent binaural impairment the formula is:
[(per cent impairment in better ear × 5) plus (per cent impairment in worse ear)] ÷ 6.

Therefore
[(0% × 5 = 0%) + 70.5%] ÷ 6 = 11.75% binaural impairment.

Please contact the Director of Research, Research Center, Subcommittee on Noise, 111 North Bonnie Brae Street, Los Angeles 27, California, for reprints or information concerning this publication.

AWARDS FOR ORIGINAL INVESTIGATION IN OTOLARYNGOLOGY

Each year the American Academy of Ophthalmology and Otolaryngology offers awards for outstanding investigative work performed during residency training. These awards are an expression of the high value which the American Academy of Ophthalmology and Otolaryngology places on original effort as the basis of medical progress.

The main purpose of the awards is to give recognition for outstanding investigations performed by young physicians who have focused their attention on Otolaryngology. The Academy also hopes that the award program will provide an extra stimulus for those who have investigative ideas as well as a stimulus to department heads to provide time and facilities so that their students can pursue these ideas.

Separate awards will be given for clinical and for basic investigations in the broad field of Otolaryngology. The broad field of Otolaryngology includes the ear, nose, throat, nasopharynx, oral cavity, larynx, esophagus, tracheobronchial tree and neck. Clinical investigation is understood to mean any study involving human patients which contributes information regarding the symptomatology, etiology, diagnosis, course, complications, therapy, etc., of any otolaryngological disorder. Basic investigation includes any study which involves histologic, physiologic, audiologic, experimental-surgical and other similar procedures or lines of investigation. Research may be both of the clinical and basic types, such as a study involving both humans and animals, and the Awards Committee will decide in which category such a manuscript is to be considered.

The competition is open to Doctors of Medicine who have completed at least one year of internship (not necessarily in the United States or Canada) and who began resident training in Otolaryngology no longer than five years past. Residency training must be in an approved American or Canadian hospital but the contestant need not be an American or Canadian citizen. The investigative work must have begun during the normal three or four year residency program but may be completed afterward. The majority of the investigative work must have been performed by the contestant, although he may acquire needed advice and technical assistance. The work need not be entirely original and may represent a continuation of research already in progress in a laboratory. The author's name should appear on the title page. Co-authors may be listed, but the contestant must be the first author. When an equal contribution to the investigation has been made by two or more qualified contestants, a letter should accompany the manuscript stating this fact. A cash award made for an entry having two or more qualified authors will be divided equally between these authors.

The Academy wishes to allow the contestant complete freedom in the publication of the manuscript. Thus, both published and unpublished papers will receive equal attention so long as they are submitted within the time limitations indicated. Reprints will be accepted, if submitted within one year of publication. Manuscripts must be typewritten and follow accepted style for journal publication. An accompanying letter should state whether the manuscript

has been published or has been submitted for publication.

Judgments of merit will be based on scientific content as well as the manner of presentation. Manuscripts should contain a minimum of historical detail, review of the literature and statements of opinion.

The manuscripts must be in the hands of Dr. Juergen Tonndorf, chairman of the Awards Subcommittee, Council Committee for Research in Otolaryngology of the American Academy of Ophthalmology and Otolaryngology not later than January 1, 1960 for awards to be presented in October 1960.

Awards will be made at the discretion of the Committee on Research and Finance of the Council of the American Academy of Ophthalmology and Otolaryngology and may be withheld if there are no manuscripts of merit. Cash awards shall not exceed six in number and shall not total more than $5000.00 in any one year. The award may be made for either clinical or basic research. Certificates of Merit may be awarded for exceptional manuscripts not winning cash awards. Letters in support of manuscripts will not be accepted by the Awards Subcommittee.

JUERGEN TONNDORF, M.D.

Chairman, Awards Subcommittee
Council Committee for
Research in Otolaryngology
American Academy of
Ophthalmology and
Otolaryngology

In Memoriam

Norma B. Elles.....Kalamazoo, Michigan
January 12, 1959

Roy Spitler Binkley.....Dayton, Ohio
November 26, 1958

William James Creighton.....Philadelphia, Pennsylvania
September 21, 1958

Ralph Allison Davis.....Chicago, Illinois
December 10, 1958

Joseph Norman Elliott.....Bloomington, Illinois
January 21, 1959

Merton A. Farnsworth.....La Junta, Colorado

Watson W. Gailey.....Bloomington, Illinois
January 19, 1959

Llewellyn E. Hetrick.....Hampton, Connecticut
January 20, 1959

Paul William Renken.....New Orleans, Louisiana
November 22, 1958

Harold Simons.....San Francisco, California
January 13, 1959

James B. Stanford.....Memphis, Tennessee
January 29, 1959

Charles Edward Walker.....Colorado Springs, Colorado
November 17, 1958

John Gill Holland.....Lynchburg, Virginia
December 24, 1958

INTERNATIONAL ASSOCIATION OF SECRETARIES OF OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETIES

The Formation of the Asia-Pacific Academy of Ophthalmology

On September 11, 1958, representatives of ophthalmologic societies from India, Ceylon, Thailand, Hong Kong, Japan, the Philippines, Taiwan, Australia, and Hawaii met in Brussels and organized the Asia-Pacific Academy of Ophthalmology.*

The purpose of this Academy was to bring together ophthalmologists practicing in Eastern Asia, the Central and Western Pacific, and Oceania. Ophthalmologic societies from countries in this region which had not sent delegates to the organizational meeting were to be informed about the newly organized Academy and invited to join.

Dr. Moacyr Alvaro, representing the Pan-American Association of Ophthalmology, and Professor Jules Francois, representing the European Society of Ophthalmology, addressed the group. They called attention to the advantages resulting from closer personal and professional contacts among ophthalmologists practicing in the same general and geographical area. They also stressed the values of postgraduate medical education available at frequent regional ophthalmologic conferences supplementing international conferences.

Pending confirmation at the Academy's next meeting, a provisional set of by-laws was adopted and a provisional slate of officers was elected. The latter includes:

President: Dr. Geminiano deOcampo, Philippine Islands

President-Elect: Dr. V. K. Chitnis, India

Vice-President: Dr. Ida Mann, Australia

Treasurer: Dr. Renald Ching, Hong Kong

Secretary-General: Dr. William John Holmes, Hawaii

Advisors:

Sir Stewart Duke-Elder, England
Dr. G. B. Bietti, Italy
Dr. Phillips Thygeson, United States
Dr. Moacyr E. Alvaro, Brazil
Dr. A. Franceschetti, Switzerland

The organizers are extending an invitation to ophthalmologists from the Americas to attend and participate in the next meeting of the Asia-Pacific Academy, to be held in Manila and Hong Kong in September 1960.

WILLIAM JOHN HOLMES, M.D.

*The formation of the Asia-Pacific Academy of Ophthalmology has been fostered by the International Association of Secretaries, which is pleased to have had a part in this undertaking which will tend to improve friendly cooperation among ophthalmologists in the Asia-Pacific area.

BOOK REVIEWS

TRANSACTIONS OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY, 1958, vol. LVI. Board, 732 pp., illustrated, plus index, University of Toronto Press, 1959.

The table of contents contains a list of the officers and council, presidents of the Society, recipients of the Howe Medal, members and associate members, necrology, minutes of the proceedings, papers, new instruments and theses. Elected at the annual meeting, May 28, 1958, were: President, Dr. Derrick Vail; Vice-President, Dr. Algernon B. Reese; Secretary-Treasurer, Dr. Maynard C. Wheeler; Editor of the Transactions, Dr. Gordon M. Bruce. Dr. Arthur Girard DeVoe was elected to the Council.

The following candidates were elected Associate Members:

Dr. Frederick C. Blodi
Dr. Leonard Christensen
Dr. Robert W. Hollenhorst
Dr. A. Edward Maumenee
Dr. Garrett L. Sullivan
Dr. Fred M. Wilson

Twenty-one papers were read on clinical, surgical and optical subjects. The publication of these contributions is not limited to the TRANSACTIONS, which has a comparatively limited circulation, and undoubtedly some of them will be published in our outstanding ophthalmic journals. However, there is an advantage in a single volume that contains the papers on contemporary presentation. Many of the papers are, in effect, additional contributions to subjects that have been in the literature for a considerable time.

A feature of the TRANSACTIONS that is unique is the publication of the accepted theses of the successful candidates. These theses are not read at any meeting of the Society and the membership knows nothing about them until they appear in the TRANSACTIONS. Candidate theses are critically scrutinized by the Thesis Committee and must represent outstanding ability of the candidate to win acceptance. They are the gems of current ophthalmic literature.

SO YOU HAVE GLAUCOMA. By Everett R. Veirs, M.D., Chief of the Section on Ophthalmology for the Scott and White Clinic, Scott and White Memorial Hospitals, and Scott, Sherwood and Brindley Foundation, Temple, Texas. Board, 64 pp., illustrated, New York and London, Grune and Stratton, 1958.

It is generally assumed that glaucoma is a blinding disease that can be controlled by medical treatment, in most cases, when detected in early stages. It is estimated that 2 per cent of persons past 40 years of age have glaucoma. As the early symptoms may not be observed until irreparable damage to sight has occurred, the wide publicity concerning glaucoma that has been given in newspapers, magazines and audiovisual facilities is fully justified. Nevertheless a certain apprehension is aroused in those whose symptoms are nonconclusive as well as those who have just been appraised of a positive indication of glaucoma by medical examination and tests.

The general reader cannot be expected to rationalize his situation without further consultation and advice. Every physician who cares for a glaucoma patient is obligated to acquaint his charge with the diagnosis and probable cause and result of the disease. This is not easy. The story must be repeated time after time to keep the patient alert to directions for treatment and to allay his

fears of the changes in symptoms and findings.

The brochure prepared by Dr. Veirs is comprehensive enough and easily understood by the average adult. Glaucoma patients refer to it frequently for reassurance and appreciate the information they need for a reasonable understanding of what is in store for them. It is recommended for all adult glaucoma patients.

FUNDUS OCULI. Ed. 3, by Hector M. Nano, Chief of the Service of Ophthalmology, Salaberry, Buenos Aires, Argentina. Board, 748 pp., illustrated. Buenos Aires, Editorial Universitaria, Oscar Macchi, 1958.

A properly illustrated atlas and text on disorders and diseases of the optic papilla and retina. Eight sections include 23 chapters based on clinical disorders, chiefly of the circulatory system of the eye. There are 305 numbered figures, many in color, from the author's collection of cases and borrowed from the literature. More than 2,000 titles are discussed under appropriate chapter headings, and an adequate index and table of contents by category facilitate location within the text. A comprehensive bibliography follows each section. Unfortunately a number of typographical errors, misspelled names, vitiates somewhat the value of the list as references.

THE MIDDLE EAR. By Heinrich G. Kobrak, M.D., Ph.D., with a foreword by John R. Lindsay, M.D. Board, 254 pp., index, $15.00. Chicago, The University of Chicago Press, 5750 Ellis Ave., Chicago 37, Ill., 1958.

Dr. Heinrich G. Kobrak was professor of otologic research at the College of Medicine, Wayne State University, until his death in November 1957. Chapters were contributed by J. E. Fournier, John R. Lindsay, Samuel Rosen, A. Wilska, H. Wullstein and Fritz Zoellner.

Treatment methods for hearing impairment caused by a diseased sound-conduction system are described by four surgeons who are intimately identified with recent advances in appropriate surgical methods. It is a valuable guidebook for physicians and surgeons concerned with the treatment of middle ear deafness.

News - - - Notes

RESEARCH IN OPHTHALMOLOGY

The National Committee for Research in Ophthalmology and Blindness, 406-C South Boulevard, Evanston, Ill., has established a confidential information exchange for groups seeking scientists, physicians or technical personnel interested in research in ophthalmology and blindness. Institutions or individuals interested may secure information from the Committee office, 406-C South Boulevard, Evanston, Ill.

The Elbyrne G. Gill Eye and Ear Foundation announces the establishment of a Research Laboratory in conjunction with the Eye Bank and Sight Conservation Society of Virginia, under the direction of Miss Jean Swartz, M. S. in biochemistry. The laboratory is prepared to make the dye test for toxoplasmosis.

MEMORIAL LECTURERS

Dr. Anderson C. Hilding, Duluth, Minn., has accepted the assignment as the Wherry Memorial Lecturer in 1960. The Jackson Memorial Lecturer in 1960 will be Dr. Lorenz Zimmerman, Washington, D. C.

EXAMINATION FOR ORTHOPTIC TECHNICIANS

The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in August and October, 1959.

The written examination will be non-assembled and will take place on Thursday, August 20, in certain designated cities. It will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, October 10, in Chicago, just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Applications for examination will be received by the office of the Chairman of Examinations, Frank D. Costenbader, M.D., 1605 22nd St. N. W., Washington 8, D. C. Each application should be accompanied by the examination fee of $30. Applications will not be accepted after July 1, 1959.

MEETINGS

West Virginia Academy of Ophthalmology and Otolaryngology

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its twelfth annual meeting, June 8 and 9, 1959, at The Greenbrier in White Sulphur Springs. On Monday, June 8, the principal speaker will be Dr. Murray McCaslin of Pittsburg, Pa. The subjects of his addresses will be "Cataract Surgery" and "Complications of Cataract." Dr. J. Warrick Thomas of Richmond, Va., will be the speaker on Tuesday, June 9. He will discuss "Headaches as Related to Allergy" and "The Diagnosis and Management of Nasal Allergies."

Oxford Ophthalmological Congress

The next meeting of the Oxford Ophthalmological Congress, to be held on July 1, 2, 3, and 4, 1959, will celebrate the fiftieth anniversary of its foundation.

The following office bearers were recently elected:
Master—Mr. O. M. Duthie (Manchester)
Deputy Master—Mr. C. H. Bamford (Derby)

Editorial Secretary—**Mr. L. P. Jameson Evans (Birmingham)**

Hon. Secretary and Treasurer—**Mr. Ian C. Fraser (Shrewsbury)**

The main discussion at the 1959 meeting will be "Ophthalmic Manifestations in Pediatric Practice." One day will be devoted to "Afterthoughts" on their Doyne Lectures delivered by fifteen past lecturers.

British Columbia Oto-Ophthalmological Conference

The 1959 British Columbia Oto-Ophthalmological Conference will be held May 28 to 30, Vancouver, B. C., Can. Alfred Huber, M.D., assistant to Professor Amsler, Zurich, Switzerland, will be the guest speaker in ophthalmology. Theo E. Walsh, M. D., St. Louis, will be the guest speaker in otolaryngology. All otolaryngologists and ophthalmologists in the Pacific Northwest are cordially invited to attend. For further information write H. B. Lockhart, M.D., Chairman, 1959 Conference, Suite 408, 1541 W. Broadway Ave., Vancouver 9, B.C., Can.

Pacific Coast Oto-Ophthalmological Society

Las Vegas, Nevada, is the site of the Pacific Coast Oto-Ophthalmological Society's annual meeting May 3 through 7. Guest speakers include R. R. Newell, M.D., Stanford University School of Medicine; William P. Mikkelsen, M.D., University of Southern California; James F. Crow, Ph.D., University of Wisconsin; Stuart C. Cullen, M.D., University of California Medical Center; and George E. Shambaugh, Jr., M.D., Northwestern University Medical School, Chicago.

Other guest speakers are Sidney Riegelman, Ph.D., University of California School of Pharmacy; Lawrence R. Boies, M.D., University of Minnesota; Dean M. Lierle, M.D., State University of Iowa; A. Edward Maumenee, M.D., Johns Hopkins University, Baltimore; John W. Henderson, M.D., Mayo Clinic, Rochester, Minn.; and Digby Leigh, M.D., Children's Hospital, Los Angeles.

The Hotel Riviera is to be the headquarters for the meeting. Write directly to the Hotel Riviera for all room reservations.

POSTGRADUATE CONFERENCES
University of Michigan Medical School

The Department of Postgraduate Medicine and the University of Michigan Medical School announces an otolaryngology conference to be held April 16, 17 and 18, 1959, in the University Hospital, Room 1603. This conference will include a review of fundamental principles and presentation of recent advances in this specialty through lectures and panel discussions by guest physicians of national prominence and members of the medical faculty at the University. The fee for the conference is $50, payable upon application. Checks should be made to The University of Michigan. The following program has been arranged.

Thursday, April 16

Surgical Anatomy of the Neck
Russell T. Woodburne, Ph.D., Professor of Anatomy

The Problem of the Dizzy Patient
David D. DeWeese, M. D., Clinical Professor of Otorhinolaryngology, University of Oregon Medical School, Portland, Oregon

Wide Field Total Laryngectomy
Walter P. Work, M. D., Assistant Clinical Professor of Surgery, Stanford University School of Medicine, San Francisco, California

The Treatment of Chronic Secretory Otitis Media
Dr. DeWeese

Functional Hearing Loss—Newer Tests for Its Detection
Dr. Work

General Principles of Wound Healing
Dr. Marion S. DeWeese

SECTION ON OPHTHALMOLOGY
AMERICAN MEDICAL ASSOCIATION
PROGRAM
June 10-12, 1959; Ambassador Hotel, Atlantic City

WEDNESDAY MORNING, JUNE 10

Clinical Aspects of Ocular Siderosis and Hemosiderosis
PAUL A. CIBIS, TSUYOSHI YAMASHITA and FRANCISCO RODRIGUES V., St. Louis
Discussion to be opened by LORENZ E. ZIMMERMAN, Washington, D. C.

Ocular Manifestations of the Chronic Renal Tubular Insufficiency Syndromes
HAROLD F. FALLS, Ann Arbor, Mich.
Discussion to be opened by ISAAC S. TASSMAN, Philadelphia

Clinical Study of Choroidal Nevi
EDWARD TAMLER, San Francisco, and A. EDWARD MAUMENEE, Baltimore
Discussion to be opened by JOSEPH A. C. WADSWORTH, New York

Embryonal Rhabdomyosarcoma of the Orbit in Children and Young Adults
WILLIAM C. FRAYER and HORATIO T. ENTERLINE, Philadelphia
Discussion to be opened by WILFRED E. FRY, Philadelphia

Mycotic Ulcerative Keratitis
SHALER ROBERTS, CAESAR GONZALEZ, ERNEST CHICK and BANKS ANDERSON, Durham, N. C.
Discussion to be opened by FRANK W. NEWELL, Chicago

Applanation Tonometry in the Diagnosis and Treatment of Glaucoma
ANDREW J. GAY and BERNARD BECKER, St. Louis
Discussion to be opened by PETER C. KRONFELD, Chicago

The Application of the Television Ophthalmoscope to Some Problems of Clinical Ophthalmology
A. M. POTTS, S. S. WEST and J. SHEARER, Cleveland
Discussion to be opened by ARTHUR LINKSZ, New York

The Effect of Alpha Chymotrypsin on the Rabbit Zonule and Lens Capsule
ROBERT H. BEDROSSIAN, Vancouver, Wash.
Discussion to be opened by RICHARD C. TROUTMAN, New York

THURSDAY MORNING, JUNE 11

EXECUTIVE SESSION

Clinical Aspects of Uveal Hypersensitivity
ROBERT S. COLES and FREDERICK H. THEODORE, New York

Long Term Steroid Therapy of Chronic Uveitis
DAN M. GORDON, New York
Discussion to be opened by IRVING H. LEOPOLD, Philadelphia

Effect of Levo-Rotary Adrenalin 2 Per Cent on the Glaucomatous Eye
LAWRENCE L. GARNER, Milwaukee; WILLIAM JOHNSTONE, Chicago; ELMER J. BALLINTINE, Cleveland; and MICHAEL CARROLL, Chicago
Discussion to be opened by WILLIS S. KNIGHTON, New York

Results of Iridencleisis in Negro and White Races
JOHN R. CASSADY, South Bend, Ind.
Discussion to be opened by SAMUEL D. MCPHERSON, JR., Durham, N. C.

Ocular Manifestations of Pituitary Tumor in Cushing's Syndrome
THOMAS P. KEARNS, ROBERT M. SALASSA, COLLIN S. MACCARTY and JAMES W. KERNOHAN, Rochester, Minn.
Discussion to be opened by FRANCIS HEED ADLER, Philadelphia

Conjunctivo Antro Rhinostomy
JAMES E. BENNETT, JAMES R. ARMSTRONG, RAYMOND E. JONES and FILLMORE SCHILLER, Cleveland
Discussion to be opened by EDMUND B. SPAETH, Philadelphia

FRIDAY MORNING, JUNE 12

PANEL ON NEURO-OPHTHALMOLOGY

Chairman's Address
FRANK B. WALSH, Baltimore

Address of Invited Foreign Guest
S. P. MEADOWS, London

Ocular Manifestations of Tumors of the Brain Stem
PAUL C. BUCY, Guest of Honor, and JAMES E. KEPLINGER, Chicago

Ophthalmological Involvement in the Primary Demyelinating Diseases
DONALD J. LYLE, Cincinnati

Some Neuro-ophthalmological Considerations in Cerebral Vascular Insufficiencies
WILLIAM F. HOYT, San Francisco

Bilateral Central Scotomas of Hysterical Origin
HARVEY A. LINCOFF, New York

See-saw Nystagmus with Suprasellar Epidermoid Tumor
J. LAWTON SMITH and VERNON H. MARK, Boston

Supratentorial Mass Lesions Presenting with Brain Stem Signs
EDWARD W. D. NORTON, Miami, Fla.

Macular Visual Fields
CHARLES J. CAMPBELL and CATHERINE RITTLER, New York

Convergence Nystagmus
DAVID G. COGAN, Boston

Panel to be discussed by S. P. MEADOWS, PAUL C. BUCY and FRANK B. WALSH

Friday, April 17

Pathologic Physiology of Cough

Nancy E. Furstenberg, M.D., Instructor in Internal Medicine

Pharmacology of Cough Suppression

Edward A. Carr, Jr., M.D., Associate Professor of Pharmacology and Internal Medicine

Treatment of Tumors of the Parotid Gland

Frank D. Lathrop, M. D., Otologist, Lahey Clinic, Boston, Massachusetts

Tympanoplasty in 3D

J. Brown Farrior, M.D., Director, Farrior Clinic, Tampa, Florida

Treatment of Facial Paralysis

Dr. Lathrop

Stapes Operations and Operability

Dr. Farrior

Current Concepts on the Use of Steroids in Medicine and Surgery

Jerome W. Conn, M. D., Professor of Internal Medicine

Saturday, April 18

Pharmacologic Aspects of the Tranquilizing Drugs

Edward F. Domino, M. D., Associate Professor of Pharmacology

Sudden Deafness

Wesley H. Bradley, M. D., Assistant Clinical Professor of Otolaryngology, University of New York College of Medicine, Syracuse, New York

Mouth Lesions

William H. Saunders, M.D., Associate Professor of Otolaryngology, Ohio State University College of Medicine, Columbus, Ohio

Further information and application blanks may be obtained from John M. Sheldon, M. D., Director, Department of Postgraduate Medicine, 1610 University Hospital, Ann Arbor, Michigan.

The Department of Postgraduate Medicine and the University of Michigan Medical School announce an ophthalmology conference to be held April 20, 21, and 22, 1959, in the Horace H. Rackham School of Graduate Studies of the University of Michigan Medical Center. This conference will include a review of fundamental principles by physicians of national prominence, as well as an opportunity to observe the application of modern developments in this specialty. Application should be made in advance. The fee for the conference is $50.00 payable upon application. Checks should be made to The University of Michigan. The following program has been arranged.

Monday, April 20

Mechanics of Intracapsular Cataract Extraction

George M. Haik, M.D., Professor of Ophthalmology and head of Department of Ophthalmology, Tulane University Medical School, New Orleans, Louisiana

The Physiological Effects of Aging on the Human Eye

Abraham L. Kornzweig, M. D., Associate Clinical Professor of Ophthalmology, New York University Postgraduate School of Medicine and Senior Assistant, New York Eye and Ear Infirmary, New York, New York

Management of Ocular Injuries (I)

Dr. Haik

Management of Ocular Injuries (II)

Dr. Haik

Development of the Angle of the Anterior Chamber in the Human Eye

Dr. Kornzweig

The Pathology of the Trabeculum in Open and Closed Angle Glaucoma and in Senescence

J. Reimer Wolter, M. D., Assistant Professor of Ophthalmology, The University of Michigan Medical School

Tuesday, April 21

Entropion and Ectropion—Senile

Sidney A. Fox, M. D., Assistant Clinical Professor of Ophthalmology, New York, New York

Lesions of the Macula in the Aged

Dr. Kornzweig

Ptosis

Dr. Fox

Corneal Surgery in Complicated Cases

Howard L. Wilder, M. D., Acting Head of Department of Ophthalmology, University of Illinois Medical School, Chicago, Illinois

Indirect Ophthalmoscopy in Daily Practice of Ophthalmology

Charles L. Schepens, M. D., Director of Retina Foundation; Clinical Associate, Harvard University Medical School, Assistant Surgeon of Ophthalmology, Massachusetts Eye and Ear Infirmary and Massachusetts General, Boston, Massachusetts.

Irregular Corneal Scleral Grafts

Dr. Wilder

Wednesday, April 22

Bilateral Retinal Detachment without Retinal Breaks. A New Disease Entity

Dr. Schepens

The High and Low Point of Lens Extraction

Dr. Wilder

Overcorrections in Muscle Surgery

Edmond L. Cooper, M. D., Instructor in Ophthalmology, Wayne University Medical School, Detroit, Michigan

Diagnosis and Conservative Treatment of Certain Intraocular Tumors

Dr. Schepens

Principles and Practice of Adrenal Corticosteriod Treatment

David H. P. Streeten, M. D., Assistant Professor of Internal Medicine, The University of Michigan Medical School

Streak Retinoscopy

Walter Z. Rundles, Sr., M. D., Flint, Michigan

On Tuesday evening the members of the conference will meet with the Detroit Ophthalmological Club, the Detroit Ophthalmological Society, and the Michigan Triological Society. At the meeting, Dr. Sidney A. Fox will deliver the annual Walter R. Parker Lecture. His subject will be "Fundamentals of Ophthalmic Plastic Surgery."

Further information and application blanks may be obtained from John M. Sheldon, M. D., Director, Department of Postgraduate Medicine, The University of Michigan Medical School, Room 1610, University Hospital, Ann Arbor, Michigan.

Akron Academy of Ophthalmology and Otolaryngology

The Akron Academy of Ophthalmology and Otolaryngology announces a postgraduate course in Allergy and Endocrinological Aspects of Allergy by Dr. Herbert J. Rinkel, Kansas City, Mo., and Dr. Z. Z. Godlowski, Associate in Medicine at Northwestern University, Chicago, May 4 to 6, 1959, at the Akron City Club, Ohio Building, Akron, Ohio. A. A. G. P. credit will be given if desired. Registration fee is $35. For further information contact A. L. Peter, M.D., 656 W. Market St., Akron 3, Ohio.

Stanford University School of Medicine

Stanford University School of Medicine will present the Annual Postgraduate Conference in Ophthalmology from Monday, April 6, through Friday, April 10, 1959. Registration will be open to physicians who limit their practice to the treatment of diseases of the eye, or the eye, ear, nose and throat. In order to allow free discussion by members of the conference, registration will be limited to thirty physicians.

Instructors will be Dr. Dohrmann K. Pischel, Dr. Jerome Bettman, Dr. Max Fine, Dr. Earle H. McBain, and Dr. Arthur Jampolsky.

Programs and further information may be obtained from the Office of the Dean, Stanford University School of Medicine, 2398 Sacramento St., San Francisco 15.

Wayne State University Postgraduate Course

The Department of Ophthalmology of Wayne State University College of Medicine will give nine months' training in basic ophthalmology beginning September 21, 1959.

Six mornings a week will be spent in lectures and laboratories. Each afternoon students will be assigned to eye clinics of affiliated hospitals. A fundus clinic will be held weekly; also, one on muscles.

Students will be given 120 hours of training in physiologic optics, 122 hours in histology and pathology, 60 hours in biochemistry, 40 hours in neuro-anatomy, and so forth.

Tuition is $300. Brochures on the basic sciences course will be mailed upon request. For further information write to A. D. Ruedemann, M.D., Chairman, Department of Ophthalmology, 690 Mullett St., Detroit 26.

POSITIONS AVAILABLE

WANTED — OTOLARYNGOLOGIST. Board certified or qualified. Busy two-man office, Long Island, N. Y., needs third associate; starting remuneration guaranteed; partnership after one year if compatible. Only those interested in permanent association need apply. For further information write Nathan Steinberg, M. D., 165 N. Village Ave., Rockville Centre, N. Y.

OPTHTHALMOLOGIST—newly equipped Reno, Nevada, office of L. V. Johnson available. Deferred payment if desired. Contact S. T. Clarke, M. D., 130 N. Virginia St., Reno, Nevada. Phone FAirview 2-4019.

California OPHTHALMOLOGIST needs associate. Financial arrangements will be left open. Further details may be obtained by writing Edwin Wortham, M.D., 1300 Bancroft Ave., San Leandro, Calif.

The University Park Medical Clinic of Denver has openings in both otolaryngology and ophthalmology. Applicants should be either Board qualified. or Board certified. Opportunities for association with the University of Colorado in research or teaching are possible. Financial arrangements are negotiable and partnership after one year a possibility. For further information, write to John W. Brown, Manager, University Park Medical Clinic, 1919 S. University Blvd., Denver 10.

Listings of positions available should be sent to W. L. Benedict, M.D., 15 Second Street S. W., Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist: diplomate, resident, etc.) (2) type of position to be filled; and (3) to whom to write for further information. Unless otherwise requested, listings will be published only once.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

FOREIGN SUBSCRIPTIONS

Some foreign members of the Academy as well as subscribers to the TRANSACTIONS are experiencing difficulty in remitting in American dollars. For their convenience the Academy will accept any legal foreign currency in payment of dues, fees for Home Study Courses, Teachers' Manuals, Orthoptic Journals, TRANSACTIONS, etc., at the prevailing rate of currency exchange.

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